DISEASES OF THE NERVOUS SYSTEM

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DISEASES OF
THE NERVOUS SYSTEM

A HANDBOOK FOR STUDENTS AND PRACTITIONERS

BY

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AND THE NATIONAL ORTHOPÆDIC HOSPITAL

WITH ILLUSTRATIONS

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PREFACE.

THE objects of this handbook are to enable students and practitioners to know how to examine patients suffering from diseases of the nervous system, and assist them to marshal their facts in a definite order, and also to serve as an introduction to the study of the larger standard works on Neurology.

For the first purpose two chapters (three and four) have been devoted to the Method of taking a Case and to the Modes of Examination employed respectively. The great difficulty of students in taking a case of nervous disease consists, as far as I have seen, in not knowing where to begin or in what order to examine the patient. As this difficulty is increased, rather than diminished, by following a regional or anatomical mode of examination, as is sometimes done, I cannot too strongly advise the importance of using in all cases, except where the cranial nerves or their nuclei are involved, the more physiological method of taking the different systems, as motor and sensory, and following each of them throughout the whole body.

The descriptions of the Anatomy and Physiology of the Nervous System are given as shortly as possible; and I have not attempted to describe the minute anatomy of the medulla and of the cranial nerve nuclei, or to give figures to illustrate the different cross-sections of this part, as the subject can be far better learnt from text-books, such as Quain's *Anatomy*, and it would be quite impossible to treat it satisfactorily in the space at my disposal.

Nearly all the illustrations in the text are original. The figures 5 and 8 are founded on the experimental researches which I made with Mr. Victor Horsley in working out more minutely the cerebral localisation of Dr. Ferrier, and which were published in the *Philosophical Transactions of the Royal Society*;[1] figure 9 is from another of our papers.[2] I am indebted to Mr. Swanzy for figures 10, 16, and 17, and to Dr. Lewis Jones for Plates II—V.

I have to tender my best thanks to my friend Dr. Head for kindly allowing me to publish his newest diagrams, Plates VI—XI, illustrating his valuable investigations on the areas of sensation supplied by

---

[1] Vol. clxxviii. (1887), B; vol. clxxix. (1888), B; vol. clxxxi. (1890), B; vol. clxxxv. (1894), B.

[2] Vol. clxxxi. (1890), B, on the Orang Outang.

the different segments of the spinal cord. These diagrams differ in some details from those he originally published in *Brain*, and the alterations are due to further investigations.

In the chapters on Disseminated Sclerosis and on Aphasia respectively, I have obtained much help from the writings of Dr. Buzzard and from those of Dr. Charlton Bastian. Though the substance of the book is founded on the clinical experience which I have had at the National Hospital, Queen Square, I must express my indebtedness to the assistance which I have received from the writings and teaching of Dr. Hughlings Jackson, and from the standard work on Diseases of the Nervous System by Sir W. Gowers.

CHARLES E. BEEVOR.

33, Harley Street, W.

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ERRATA.

Page 193, three lines from bottom of page, for "convolution" read "lobe."

Page 207, third and fourth lines from top, the words "by a lesion" should come before the word "anywhere."

Page 269, seventh line from top, omit the word "even."

Page 405, thirteenth line from top, the words "the intercostals" should come after the word "pharynx."

DISEASES OF THE NERVOUS SYSTEM.

CHAPTER I.

INTRODUCTORY.

It will not be possible in this work to give anything like a complete account of the Anatomy and Physiology of the Nervous System, and for this purpose standard works such as Quain's *Anatomy* and Foster's *Physiology* must be consulted. A brief sketch of the nervous system, and especially those parts which are affected by disease, will, however, be advantageous.

The simplest example of the mechanism of the nervous system is that presented by what is known as reflex action. As an illustration of this action we may take the plantar reflex; here the sole of the foot is tickled by a quill pen, and in a very short interval the foot is drawn up away from the irritating body. To produce this effect the nervous apparatus must consist of the following parts (fig. 13):—

1. An afferent sensory nerve fibre (*s.n.*) having an end organ in the skin (s.) capable of appreciating and transmitting the impression of the quill pen.

2. A central station (*a.h.c.*), in which this impression is received, and where it can be transformed into a motor excitation.

3. An efferent motor nerve (*m.n.*), which carries the motor excitation down to the muscle (M.), or group of muscles, and causes them to contract and produce a certain movement.

The same kind of action takes place in the case of the conjunctival reflex; the conjunctiva is touched, and is followed by the contraction of the orbicularis palpebrarum, producing a blinking of the eyelids.

When we examine the structures which take part in these reflexes, we find in the case of the plantar reflex that—

1. The afferent sensory nerve, which has its end organ in the sole of the foot, when traced upwards, passes into the spinal cord by the posterior root of the first sacral nerve.

2. The central station is the grey matter of the spinal cord, at the level of the first three sacral nerves, where the sensory impression is received, and acts on the large motor cells of the anterior horn of the grey matter.

3. The efferent motor nerves, which carry the motor excitation from the motor cell down to the muscles, are the anterior roots of the upper sacral nerves, which produce a contraction in the flexor muscles of the ankle and knee, and so withdraw the sole of the foot from the seat of irritation.

These three together make up a reflex arc.

In the same way with the conjunctival reflex, the afferent sensory fibres are carried up by the fifth cranial nerve to the central station, which is in the grey matter of the medulla; and the efferent motor nerve is the seventh cranial nerve.

Now, although the plantar reflex is quite independent of consciousness and of volition and can take place during sleep or during the anæsthesia of chloroform or ether (if not too profound), each reflex action is attended by the perception by the individual of the particular stimulus; and further, in many cases in the normal condition, he is able to bring other muscles into action, and so prevent the reflex act taking place.

The nervous apparatus which is required to carry out this further action is (fig. 13)—

1. An afferent system of sensory fibres (*p.c.*, *p.c.c.*, *l.*), which can conduct the impression up to a central station.

2. A central station or stations (*c.s.*, *c.m.*) of a higher degree of organisation than the grey matter of the spinal cord, where the character and nature of the impression, and the part of the body from which it came, can be recognised. In these stations also is determined whether any notice shall be taken of the sensory impression, and from them motor excitations originate.

3. An efferent system of fibres (*p.t.*, *l.c.*), which pass down to a lower central station of the spinal cord, similar to that of the reflex arc, and can prevent or neutralise the movement of the muscles, by transmitting the motor excitations from the higher central station.

In the case of the plantar reflex the sensory impression, which enters by the posterior root, is conducted up the spinal cord in the white columns, and probably by relays of fibres through the medulla and pons, to the central station—*viz.* the grey matter of the brain cortex of the opposite side; and here the character of the stimulus is recognised, and also the part of the body—the sole of the foot—whence it originated. In the grey matter of the brain is determined whether the reflex action shall be allowed to take place or not; and if not, that part of the brain in which the different movements of the body are represented is stimulated, and particularly that part where the movement is represented which will neutralise the reflex action. This motor excitation is carried down from the brain by an efferent system of fibres known as the pyramidal tract, which extends down to the neighbourhood of the motor cells of the grey matter of the spinal cord at the level of the upper sacral nerves. The motor cells which supply the extensors of the knee and ankle are thus excited, and by the contraction of these muscles the foot is prevented from being drawn up. Opinions differ as to whether the part of the cortex of the brain where the afferent fibres end is the same as the part where the efferent motor impulses start; but it is probable that, as will be shown later, they are distinct.

Reflex action is the only nervous mechanism in infancy; but as the brain becomes developed, the power to call in the aid of other muscles and prevent the reflex act becomes possible.

In some reflex actions, such as the sexual act, although there is a marked sensory perception, the individual has no power to prevent the action taking place; this is owing to there being no arrangement of muscles which could be made to act to prevent or neutralise the reflex action. In the case of the pupil reflex to light there is no perception of the contraction to the individual, and no power to prevent it taking place.

Reflex action is the simplest form of a nervous action. As an example of a complex nervous action we may consider the process of learning a musical instrument, as the piano or violin. Here the impressions from without—the example and precept of the teacher and the printed notes—come through the auditory or visual end organs, and are transmitted by the afferent optic or auditory nerves to the central stations, the grey matter of the brain. These "sensory stations" are in communication with the (motor) cortex of the brain where the actions of the different muscles are represented, and those parts, where the particular movement to be performed is represented, are excited, and thence an impulse is sent down the efferent tract to the lower central station in the brachial enlargement of the spinal cord, and the required muscles are put into action.

At first it takes some time and consideration for the co-ordination of the different parts of the brain where these muscles are represented; but by practice the arrangement becomes more quickly made, till the muscular movement follows directly on the visual impression of the printed notes—the movement becomes automatic, and is performed without any thought or consideration.

It will thus be seen that all nervous processes are sensori-motor, and that motor actions are stimulated by sensory impressions received at the time, or by the revival of past sensory impressions received at some previous period. It will therefore be necessary to describe in the nervous system afferent fibres, central stations, and efferent fibres.

CHAPTER II.

GENERAL ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM.

THE cerebro-spinal nervous system may be considered anatomically, omitting the cerebellum and the basal ganglia, as consisting of the *cortex* or outer grey matter of the two hemispheres of the brain, which are joined together by commissures, of which the corpus callosum is the most important; of the *corona radiata* or fan-shaped arrangement of white fibres, which, starting from all parts of this cortex, converge through the *internal capsule* to the *crus cerebri* or peduncle of the brain, whence they are continued down the *medulla* and *spinal cord*, and are in relation there with the cells of the grey matter. Connected with this grey matter are the *cranial* and *spinal nerves*, which thus keep in communication with the brain all parts of the body, and by which all sensations from without are conveyed up to the brain, and all impulses sent out from the brain can act upon the muscles. Following, however, the scheme sketched in the previous chapter, the different parts will be described under these headings: 1. Peripheral nerves; 2. The spinal cord; 3. The pons and medulla; 4. The internal capsule; 5. The cortex cerebri.

1. **The peripheral nerves.**—Under this heading are

included the nerves of the limbs which come from the brachial plexus, as the median, ulnar, and musculo-spiral nerves, and those from the lumbar and sacral plexuses, as the anterior crural and great sciatic. With regard to their functions, it must be remembered that most of them are mixed nerves—*i.e.* motor and sensory; and that when the functions of a nerve are completely lost—*e.g.* cut across—sensation is lost in that part of the skin, and the power of contraction in the muscles, supplied by it. The sensory areas of the skin supplied by the different peripheral nerves of the upper and lower limbs are given at the end of the book.

The *structure* of a peripheral nerve consists of a central axis cylinder—which is the continuation of the axis cylinder process of a nerve cell—surrounded by the medullary sheath or white substance of Schwann, composed of myelin, a fatty substance, which is interrupted at regular intervals by nodes, and each internodal part contains a nucleus. Outside this substance is the neurilemma or primitive sheath. This is the composition of a medullated nerve fibre. In the non-medullated fibres the medullary sheath is absent. The chief physiological importance about the nerves is that the axis cylinder is the prolongation of the chief process or axis cylinder process of a cell, whether this be situated in the cortex cerebri, the grey matter of the spinal cord, or in the ganglion on the posterior spinal root. The chief pathological importance is that, when this cell is destroyed or damaged, the nerve thus connected with it undergoes degeneration along its whole course, and also that, when a nerve is severed from its cell, the part thus separated undergoes degeneration.

This is called "Wallerian degeneration," from the name of its discoverer.

According to Ranvier the first change in the degeneration, which occurs twenty-four hours after dividing a nerve, is an increase in the size of the nuclei and the protoplasm round it, which compresses the myelin sheath, and at last separates the myelin and the axis cylinder into two parts. The nuclei divide and subdivide, and the myelin is further broken up into globules, which are removed by the lymphatics till the sheath is empty. The interruption in the nerve conduction occurs about the second day, and about this time electrical excitation is lost, while complete destruction takes place in about two weeks. Regeneration of a nerve may occur as long as its cell is intact, and it consists in a growing out of the axis cylinder from the end connected with the cell, and this gets covered up with a fresh myelin sheath.

When followed upwards, the chief nerves of the limb are traced to the brachial and lumbo-sacral plexuses, which are made up of the spinal nerves.

The brachial plexus is formed from the anterior divisions of the fourth, fifth, sixth, seventh, and eighth cervical and the first dorsal nerves; the lumbar plexus from the anterior primary divisions of the upper four lumbar nerves, with sometimes a branch from the twelfth dorsal; while the sacral is formed by the fourth and fifth lumbar and the four upper sacral nerves.

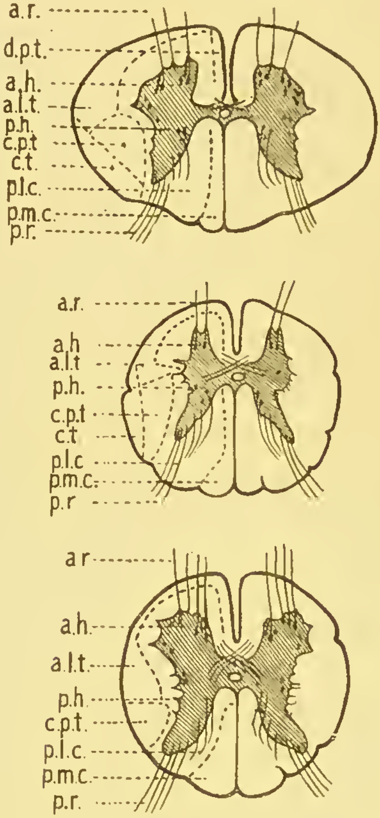
Each spinal nerve springs from the cord by two roots, an anterior motor, and a posterior—the larger—sensory with a ganglion. The two roots join beyond the ganglion, and form a motor-sensory nerve, which divides into a large anterior primary division and a small posterior.

In the table at the end of the book are given the muscles which are supplied by the motor fibres, and diagrams of the areas of the skin supplied by the sensory fibres of the spinal cord for the level of each spinal root; it is possible, but not certain, that the roots have the same motor and sensory distribution as the part of the cord from which they spring. It must be particularly noted that the motor and sensory distribution supplied by each segment of the cord is entirely different from that of the peripheral nerves.

2. The **spinal cord** consists of two symmetrical halves joined by a commissure (figs. 1, 2, 3). Behind this commissure the two halves are joined by the posterior median septum, and in front of the commissure the two halves are separated by the anterior median fissure. Each half contains a central grey matter forming two horns,—an anterior, which is most developed at the brachial and lumbar enlargements, and contains the large cells considered to be motor, and is in connection with the anterior or motor roots; and a posterior horn, in connection with the posterior or sensory root, and containing from the seventh cervical to the third lumbar roots a collection of bi-polar cells called Clarke's column, which are probably in communication with the posterior roots peripherally, and centrally with the cerebellar tract (Mott). The grey matter in the two halves of the cord is joined by a grey commissure, in the middle of which is the central canal. In front of this is the white commissure connecting the anterior columns. The grey matter is composed of a spongy

substance, in which are the large nerve cells and a fine network of medullated fibres supported in a neuroglia.

Surrounding the grey matter are the white medullated fibres, supported by the connective tissue which is sent in as septa from the pia mater investing



Figs. 1, 2, 3.—Horizontal sections of the cervical enlargement, mid-dorsal region, and lumbar enlargement of the spinal cord of man. *p.r.*, posterior root; *p.m.c.*, posterior median column; *p.l.c.*, posterior lateral column; *c.t.*, cerebellar tract; *c.p.t.*, crossed pyramidal tract; *p.h.*, posterior horn; *a.l.t.*, antero-lateral tract; *a.h.*, anterior horn; *d.p.t.*, direct pyramidal tract; *a.r.*, anterior root.

the cord, and conveys the blood vessels. The fibres. have no neurilemma; but they are supported by the neuroglia, which consists of a fine network of connective-tissue fibres in a granular matrix with nucleated cells, called glia cells or "Deiters' cells"; the fibres are arranged vertically and in columns, which have different functions.

Beginning posteriorly at the middle line, we have in a horizontal section, the posterior median column (or Goll's column), separated from its fellow of the opposite side by the posterior median septum, and on its outer side by a small septum from the postero-external column (or Burdach's column); the latter is bounded externally by the posterior horn, and through it the posterior roots pass to reach the inner side of the posterior horn. Outside the posterior horn we have the lateral column, the continuation downwards of the pyramidal decussation; and quite anteriorly next to the anterior median fissure is the direct pyramidal tract, the continuation downwards of the pyramidal tract which does not decussate. The crossed pyramidal tract does not quite extend to the surface of the cord, and on horizontal section it is found to be separated, in cases where there is degeneration of this tract, by a narrow band of fibres known as the direct cerebellar tract, which passes up through the restiform body to the cerebellum; anterior to this tract is the antero-lateral ascending tract of Gowers.

The pyramidal tracts, crossed and uncrossed, are in communication with the cells of the anterior horns the whole way down the cord, and from these cells issue the anterior root nerves which supply the muscles; these cells, like those of the motor nuclei of the medulla, exert a trophic influence on the muscles, so that if the cells are destroyed the muscles atrophy, degenerate, and show marked electrical changes (see p. 72). The cells of the anterior horns and the anterior roots in connection with them supply definite groups of muscles; this is especially the case for the cervical and lumbar enlargements for the supply of the limbs (see table at end of book). On horizontal section the cells are seen to form three groups—the mesial, the ventro-lateral, and the dorso-lateral, the last being mainly developed in the brachial and lumbar enlargements.

3. **Pons and medulla.**—At the lowest end of the medulla the posterior columns of the spinal cord end in two bundles, the funiculus gracilis and the funiculus cuneatus, which contain grey matter, the nucleus gracilis and the nucleus cuneatus, from which fibres pass upwards and cross by the internal arcuate fibres to the opposite side, decussating with their fellows of the other side, and pass into the column of fibres called the lemniscus or fillet, situated at the posterior part of the pons and medulla. Part of the lemniscus passes up by the crusta of the crus cerebri, and probably to the posterior (sensory) part of the internal capsule (fig. 11).

The afferent sensory nerve of this region is the fifth or trigeminal nerve, and just before it enters the middle of the pons on one side it has on its trunk the Gasserian ganglion.

The excitable (or motor) part of the internal capsule is continued downwards through the crusta of the crus cerebri to the anterior region of the pons and medulla, where it forms a longitudinal bundle of fibres called the pyramidal tract; at the lowest end of the medulla a small part of this tract is continued direct into the anterior columns of the cord, but most of it decussates with its fellow of the opposite side into the opposite

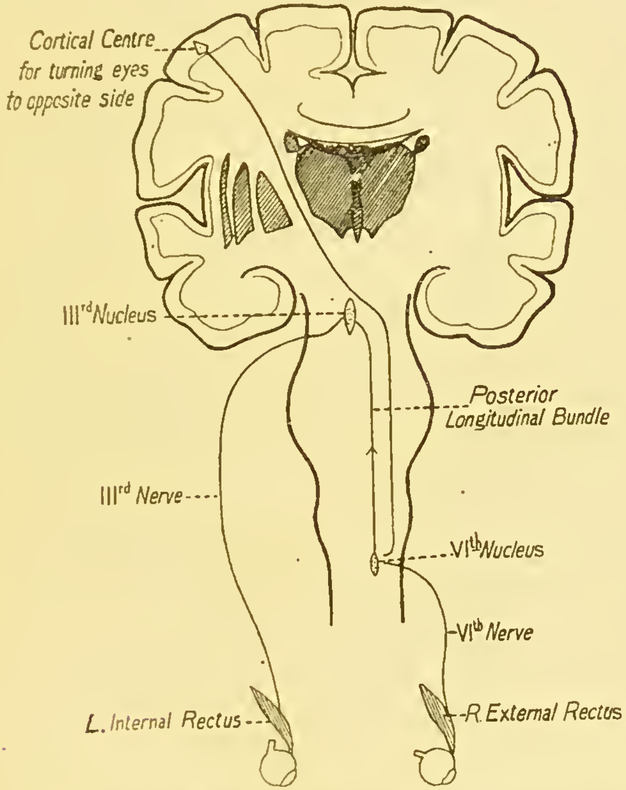


Fig. 4.—Diagram showing the effect of *stimulation* of the left cortical centre for turning the eyes laterally. The motor impulse goes from the left cortex through the left corona radiata and internal capsule to the right sixth nucleus, from there it passes up to the left third nucleus; stimulating the R. external rectus and the L. internal rectus, and turning both eyes to the right. In *paralysis* of the left cortical centre, internal capsule, or right sixth nucleus, the converse occurs, and the eyes deviate conjugately to the left.

lateral column of the spinal cord, forming the decussation of the pyramidal fibres. During this course through the pons and medulla, fibres are sent from the pyramidal tract to the nuclei of the motor cranial nerves of the opposite side mainly.

Of the motor nuclei in the pons and medulla those of the third and fourth nerves and the sixth nerve are for the muscles of the eyeball. Of the movements of the eyeballs the most important is the lateral, and fibres from the pyramidal tract are considered to cross over to the opposite sixth nucleus (fig. 4). In connection with the sixth nucleus (through the posterior longitudinal bundle) is the part of the third nucleus of the same side as the pyramidal tract, which supplies the internal rectus muscle; so that with a lesion of the *right* sixth nucleus we have paralysis of the right external rectus and of the left internal rectus, and consequently conjugate deviation of both eyes to the *left*. The same paralysis would be caused by a lesion of the left pyramidal tract above the point of crossing of its fibres to the right sixth nucleus, also by a lesion of the left internal capsule or cortex. Therefore in a lesion of the capsule or cortex the patient's eyes are directed *towards* the lesion, and in the case of the sixth nucleus *away* from the lesion.

The fifth nucleus supplies the muscles for mastication, the seventh those of the face, the eleventh nerve—the accessory to the vagus—sends motor branches to the soft palate and larynx, and the twelfth to the tongue. Each nucleus—right or left—supplies only the muscles of its side—*e.g.* one vocal cord, and one half of the palate.

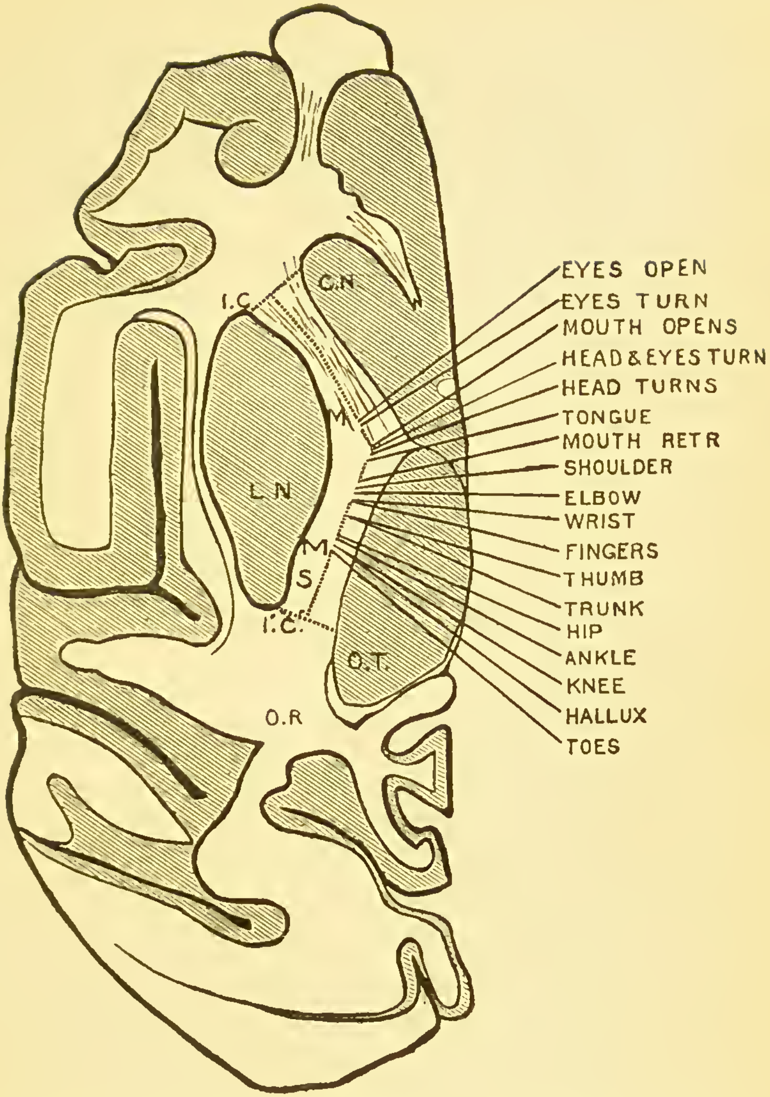


Fig. 5.—Horizontal section of the left hemisphere of the brain of a monkey to show the excitable area of the internal capsule; the movements of the different parts of the body and the order in which they were obtained when the fibres were stimulated electrically are shown in the margin. C.N., caudate nucleus, and L.N., lenticular nucleus, of the corpus striatum; O.T., optic thalamus; I.C., I′.C′., internal capsule; M.—M., excitable or motor part; S., sensory part; O.R., optic radiations.

There is reason to believe that the motor fibres of the orbicularis palpebrarum and of the orbicularis oris in the facial nerve really come from the third nucleus (Mendel) and the hypoglossal nucleus (Gowers) respectively, so that only the middle face muscles are supplied by the facial nucleus itself.

4. **Internal capsule.**—This is the part of the white fibres which is included between the basal ganglia (corpus striatum and optic thalamus). In horizontal section of the cerebral hemisphere it consists of two limbs joined at an angle or genu. At the level shown by the diagram (fig. 5) the anterior limb is not excitable electrically, except at its posterior part, and contains the fibres, having a horizontal direction, from the anterior non-excitable area of the cortex ; the posterior limb is excitable for the anterior two-thirds, and contains the fibres (here cut transversely) coming down from the excitable area of the cortex; the functions of these fibres and the movements obtained by electrical excitation, as ascertained experimentally by Mr. Horsley and the writer,[1] are shown in fig. 5. This is the part which is most commonly affected in hemiplegia. The posterior third of the posterior limb is non-excitable, and contains fibres for the conveyance of sensory impressions upwards to the cortex; in the white matter posterior to the internal capsule are fibres for sight (hemianopia, p. 30), hearing, and perhaps smell and taste for the opposite half of the whole body; this region has been called the sensory crossway. The internal capsule is continued upwards to the cortex as the corona radiata, and downwards into the crusta of the crus cerebri.

5. **Cortex.**—(1) *Anatomy.* The grey matter covers

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[1] *Phil. Trans.*, B, vol. clxxxi., 1890.

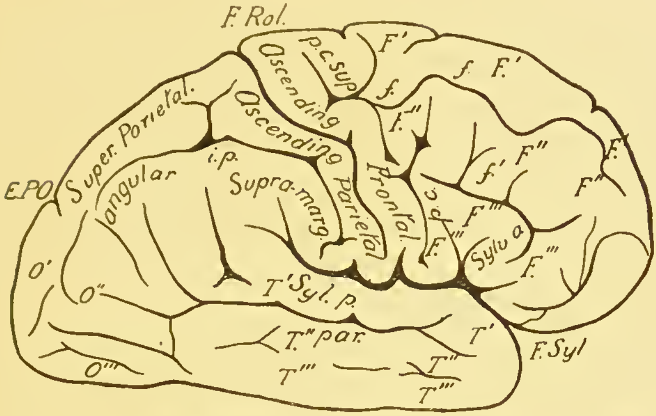

Fig. 6.—External surface of right hemisphere of human brain. Fissures: *F.Syl.*, Sylvian; *Syl.a.*, *Syl.p.*, its anterior and posterior limbs; *F.Rol.*, fissure of Rolando; *E.P.O.*, external parieto-occipital. Sulci: *p.c.*, inferior præcentral; *p.c.sup.*, its superior portion; *i.p.*, intra-parietal; *par.*, parallel; *f.*, *f'*., superior and inferior frontal. Convolutions: *F'*, *F''*, *F'''*, the three frontal; *T'*, *T''*, *T'''*, the three temporo-sphenoidal; *O'*, *O''*, *O'''*, the three occipital. The others are indicated by their names.

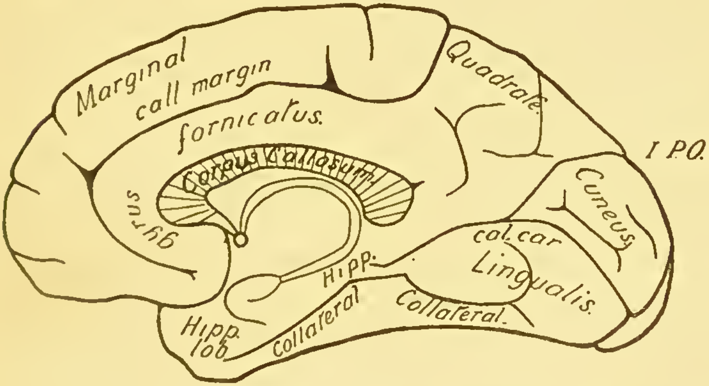

Fig. 7.—Internal surface of right hemisphere of human brain. Fissures: *call. margin.*, calloso-marginal; *calcar.*, calcarine; *I.P.O.*, internal parieto-occipital. Convolutions: *Hipp.*, hippocampal; *Hipp. lob* hippocampal lobule. The others are indicated by their names.

the whole outer surface of each hemisphere as convolutions, and consists of layers of cells, usually described as five in number, of which the third layer contains the large pyramidal cells. These cells are most numerous in the excitable part of the cortex, and are considered to be motor. With regard to the anatomy, we observe (figs. 6 and 7) that there are on the outer surface of each hemisphere—

Three fissures[1] — *i.e.* Sylvian, Rolandic, parieto-occipital (very small)—dividing the cortex into the four lobes—frontal, parietal, occipital, and temporo-sphenoidal.

Three sulci of importance, dividing up these lobes — *i.e.* præcentral, intra-parietal, and parallel (so called because parallel to and below the fissure of Sylvius).

The fissure of Sylvius is the most important and conspicuous fissure. Starting from the base of the brain, and winding outwards to the external surface of the hemisphere, it gives off a short anterior ascending limb and a posterior horizontal limb.

The convolutions are best understood, not by beginning with the extreme frontal region, but by taking the fissure of Rolando as a starting-point. If the median edge of the outer surface of the hemisphere be followed from before back, this fissure is the first definite one which can be traced downwards and forwards, making an angle of about 67° with the median line. The convolution immediately in front of this fissure is the ascending frontal, the convolution behind it is the ascending parietal, and they run from the median line down to the fissure of Sylvius. The former convolution is bounded anteriorly by the superior and inferior divisions of the præcentral sulcus; in front of this sulcus there are the three frontal convolutions—superior, middle, and inferior—the middle one being separated by the superior and inferior frontal sulci from the other two. The ascending parietal convolution is bounded posteriorly by the intra-parietal sulcus, and it is continued above into the parietal lobule or superior parietal convolution. Below and behind the intra-parietal sulcus are two more convolutions—the supra-marginal around the posterior end of the fissure of Sylvius, and the angular around the posterior end of the parallel sulcus. In the occipital lobe there are three convolutions, often not well defined; and in the temporo-sphenoidal lobe three convolutions, the first and second of which are separated by the parallel sulcus. It will thus be seen that, after taking away the ascending frontal and parietal convolutions, each lobe has *three* convolutions; and this is also the number of the chief fissures on the outer surface of each hemisphere, and there are *six* sulci.

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[1] The space between the two median surfaces of the hemispheres is called the longitudinal fissure.

The median surface of each hemisphere is not so important as the outer surface, and here again we have *three* fissures:—

The *calloso-marginal*, beginning at the median line just behind the Rolandic fissure, and running forwards and round the front of the corpus callosum.

The *calcarine*, beginning below the posterior end of the corpus callosum, and running backwards into the occipital lobe.

The *internal parieto-occipital,*[1] a continuation of the external parieto-occipital ; it runs into the calcarine.

Of the convolutions the most important are the marginal, above the calloso-marginal fissure, and the gyrus fornicatus below it; this convolution, traced backwards behind and below the corpus callosum, is continued into the gyrus hippocampi along the median surface of the temporo-sphenoidal lobe, and at the extreme anterior end of this lobe it forms the hippo-

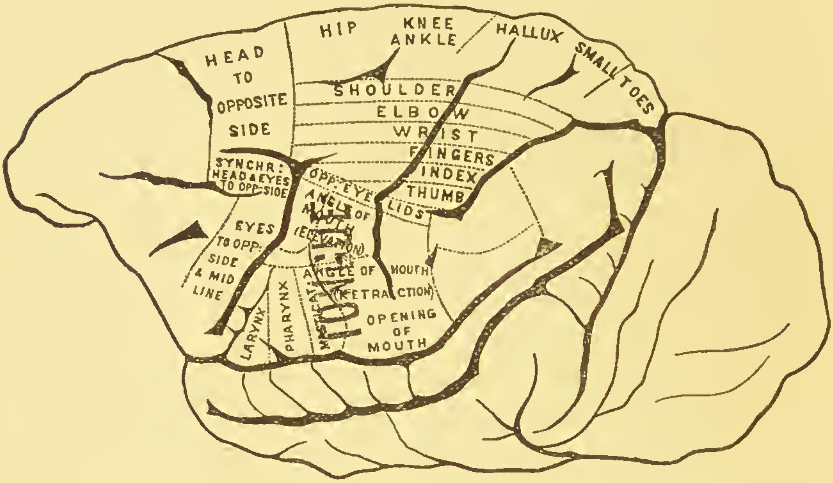

Fig. 8.—External surface of left hemisphere of monkey's brain, showing the maximum representations of the different movements obtained by electrical stimulation. The Sylvian fissure bounds the excitable area below ; the Rolandic fissure extends from "HALLUX" to "OPENING OF MOUTH." The intra-parietal sulcus bounds posteriorly the upper part of the area, and the præcentral sulcus extends from "OPP(osite) EYELIDS" above to in front of "LARYNX" below, while the superior præcentral sulcus, separating the localisation of the movements of the upper and lower limbs, is just above "SHOULDER."

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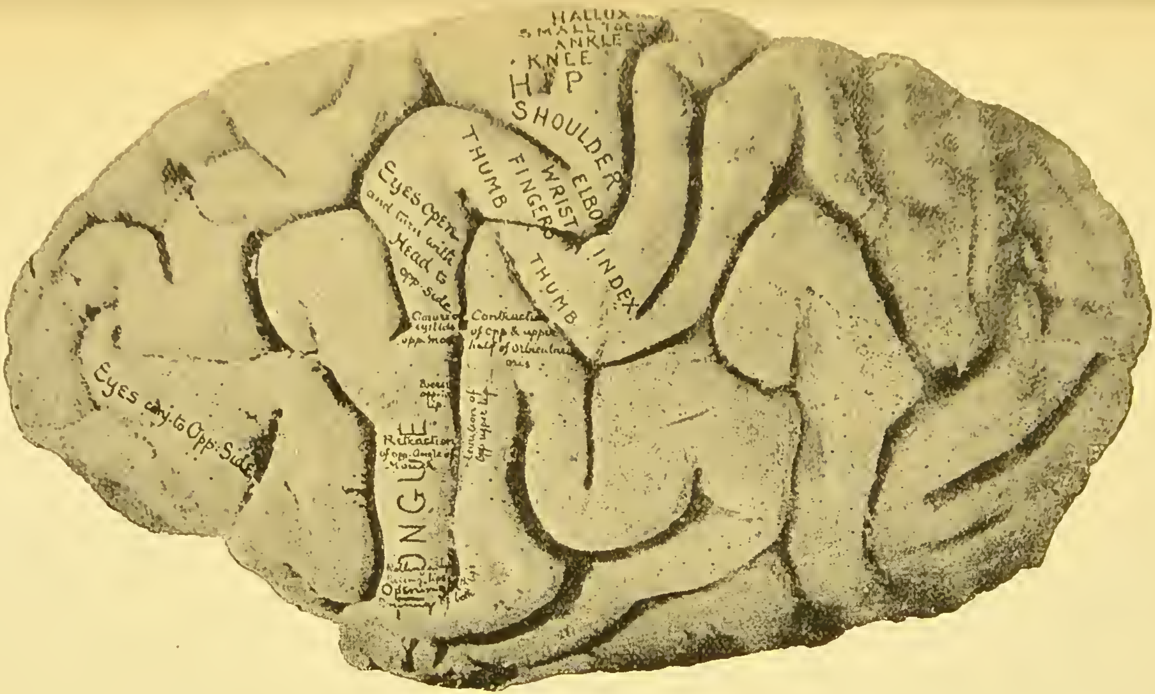
[1] The collateral is sometimes described as the sixth fissure, the internal and external parieto-occipital fissures being counted as one fissure.



HALLUX
SMALL TOES
ANKLE
KNEE
HIP
SHOULDER
ELBOW
WRIST
FINGERS
THUMB
INDEX
THUMB
Eyes open and turn with Head to opp. side
Contraction of opp & upper half of Orbicularis oris
Retraction of opp. Angle of Mouth
TONGUE
Opening of Mouth
Eyes chy. to Opp. Side

Temporo-sphenoidal Lobe.

Fig. 9.—External surface of left hemisphere of the brain of the orang outang, tilted towards the observer, so as to show the part between the Sylvian fissure and the middle line. The movements obtained by electrical stimulation and their localisation are shown by the names, and they are represented chiefly on the ascending frontal convolution, which is separated from the ascending parietal by the Z-shaped fissure of Rolando. The posterior part of the Sylvian fissure is seen, behind the inferior end of the Rolandic fissure, as a tri-radiate fissure. Note the representation of the hallux at the upper end of the ascending frontal and anterior to the Rolandic fissure, and of the thumb just above the genu of the Rolandic fissure, and above the præcentral sulcus.

campal lobule. The cuneus is the convolution between the internal parieto-occipital and calcarine fissures, while the lingualis lies below the calcarine.

(2) *Functions of the cortex cerebri.*—These will be best understood by dividing the cortex into—(*a*) a part which is excitable, *i.e.* which when stimulated by a weak faradic current gives rise to muscular movements in different parts of the body; (*b*) a part which is practically non-excitable.

(*a*) In the monkey (fig. 8) the excitable part includes on the outer surface the ascending frontal and parietal convolutions on either side of the fissure of Rolando, the parietal lobule, the posterior third of the three frontal convolutions, and on the median surface the contiguous parts of the marginal convolution; while in the orang (fig. 9) it occupies chiefly the ascending frontal and parietal.

The movements thus obtained may be arranged, according to the researches of Mr. Horsley and the writer, in the following order, beginning with the trunk muscles in the marginal convolution (Horsley and Schäfer), and passing along the ascending frontal and parietal convolutions down the fissure of Rolando, and on reaching the Sylvian fissure passing forwards. The representation here given is that which was found in the orang, supplemented by that which was obtained in the monkey; and it is highly probable that this is the arrangement which exists in man.

Muscles of the trunk.
Lower limb—Great toe.
            Small toes.
            Ankle.
            Knee.
            Hip.

Upper limb—Shoulder.
Elbow.
Wrist.
Fingers.
Thumb.
Upper part of face—Opposite eyelids, closed.
Lower part of face—Elevation of the upper lip.
Retraction of the angle of the mouth.
Opening of the mouth and rhythmical movements of mastication.
Tongue.
Vocal cords.

The difficulty of remembering this order will be much facilitated by noting that the representation of the movements are arranged from above downwards from the most coarse movements of the muscles of the trunk in the marginal convolution through the less coarse movement of the lower limb and the more complex movements of the arm and hand, to the most complex and highly specialised movements of the face, and at the anterior part next to the Sylvian fissure culminate in those of the vocal cords (Semon and Horsley); while in man this part of the cortex for the representation of the vocal cords is close to the centre for speech, situated in the left third frontal convolution.

In addition to the above movements, there are the important ones of turning the head and eyes to the opposite side, represented especially in the posterior third of the three frontal convolutions.

There is no hard-and-fast line between the different parts of the cortex for the representation of the leg, arm, and face; but a line drawn horizontally from the superior præcentral sulcus between the first and second frontal convolutions backwards through the fissure of Rolando to the intra-parietal sulcus would separate the cortex for the representation of the lower limb from that for the upper limb; while a line from the angle of the præcentral sulcus drawn backwards to the bend—convex forwards—in the fissure of Rolando would divide the cortex where the upper limb is represented from that in which the face is. This bend or genu of the Rolandic fissure in man (fig. 6) is a little nearer to the Sylvian fissure than half-way between this fissure and the median line, so that between the genu and the median line the upper half is for the representation of the leg, the lower is for the arm, and from the genu to the fissure of Sylvius for the face.

In the above list the movements of the different joints were produced by electrical stimulation of the cortex, and for the most part the movements were obtained on the opposite half of the body only—*i.e.* the representation was unilateral;[1] in fact, all the movements were unilateral except the following, which were bilaterally represented—*i.e.* the muscles on both sides simultaneously contracted when either cortex was stimulated, *viz.* opening the eyes, opening the mouth, movements of mastication, screwing up the mouth as in whistling, vocal cords, and some of the movements of the tongue, as protrusion and retraction straight. It used to be taught that each half of the tongue was acted upon independently by the cortex of the opposite hemisphere, but it is quite certain that the two halves of the tongue are thrown into action by stimulating one cortex only. The most important unilaterally represented movement of the tongue is that of protrusion to the opposite side—*i.e.* stimulation of the left cortex protrudes the tongue to the right, both halves of the tongue are in action, the left half being advanced while the right is retracted.

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[1] The exception to this rule is the action of the sterno-mastoid in rotating the head to the opposite side, for on stimulating the *left* cortex, although the head turns to the *right* acting in harmony with the right hand, it is the *left* sterno-mastoid which is thrown into action by the left cortex, as it is a mechanical convenience that the muscle is not on the side towards which the movement is required.

(*b*) The non-excitable cortex consists of the part in front of the excitable area, comprising the anterior two-thirds of the three frontal convolutions, and the part behind the excitable area, containing the rest of the parietal, the occipital, and the temporo-sphenoidal lobes.[1]

The part of the cortex in front of the excitable area is considered to be the seat of the highest intellectual processes.

In the part behind the excitable area, the angular gyrus is considered to be a portion of the visual centre, and a lesion of it causes word blindness.

In the posterior part of the first temporo-sphenoidal convolution is the centre for hearing for the opposite ear, and a lesion of it causes word deafness.

The extreme anterior part of the temporo-sphenoidal lobe (the hippocampal lobule) is considered to be the seat of taste and smell for the opposite side.

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[1] It should be remarked that some of this posterior part has been found to be excitable, slight movements of the eyes having been obtained by excitation of the occipital region, but having a different and more feeble quality than that obtained from the "excitable area."

On the median surface of the occipital lobe, and particularly in the cuneus, the convolution above the calcarine fissure, and in the lingual gyrus below it, are the centres for the localisation of sight, and lesion of these parts produces hemianopia—*i.e.* a lesion of the left occipital region produces inability to see objects in the right field of vision in either eye.

With regard to the localisation of sensation in the cortex it is very difficult to speak definitely, but there seems no doubt that removal of a part of the excitable (motor) cortex produces blunting of sensation, a difficulty to localise correctly, and loss of muscular sense in the limb supplied by this removed cortex (Horsley). The localisation for complete loss of sensation is placed by some observers in the gyrus fornicatus and gyrus hippocampi, by others in the parietal lobe.

Besides the cortex of the brain and the corona radiata, there are in each hemisphere spaces or ventricles formed during the development of the brain; masses of grey matter forming the basal ganglia; and the tracts of fibres which connect the end organs of the special senses with the cerebral cortex to which they convey the sensory impulses received from without.

Ventricles.—There are two lateral ventricles, one in each hemisphere extending horizontally for nearly the whole length of the hemisphere, below the corpus callosum and on the inner side of the internal capsule, while the floor is formed by the basal ganglia—the caudate nucleus and optic thalamus. Each ventricle has a descending cornu into the temporo-sphenoidal lobe.

Each ventricle is connected with its fellow and with the third ventricle by the foramen of Monro. The third ventricle lies between the optic thalami, and is connected behind by the "iter" or Sylvian aqueduct with the fourth ventricle, a lozenge-shaped space; this is continued downwards into the central canal of the cord, of which the ventricle is the expansion. The fourth ventricle is bounded in front by the posterior surface of the medulla and pons, and behind by the valve of Vieussens and the cerebellum; on the floor near the lower end of the space the eminences formed by the nuclei of the vagus and hypoglossal nerves are seen.

The **basal ganglia**—corpus striatum and optic thalamus—are seen in horizontal and vertical sections through the hemispheres (figs. 5, 11, 12). The two parts of the corpus striatum bound the anterior limb of the internal capsule, the caudate nucleus being on the inner and the lenticular nucleus on the outer side, while the posterior limb is bounded by the lenticular nucleus on the outer side and the optic thalamus on the inner. They are composed of cells with a fine network of fibres. The function of the caudate nucleus is at present unknown, and it is inexcitable by electrical stimulation; the optic thalamus is probably associated with vision.

The course of the **visual fibres** is very important, and it can be best understood by tracing the path from the object in the field of vision to the cortex cerebri. The whole field of vision in the horizontal direction is half a circle or 180°, while in the vertical meridian the angle is rather less than 180°, owing to the projection of the forehead. Horizontally each eye cannot see 180°, owing to the nose getting in the way of the rays

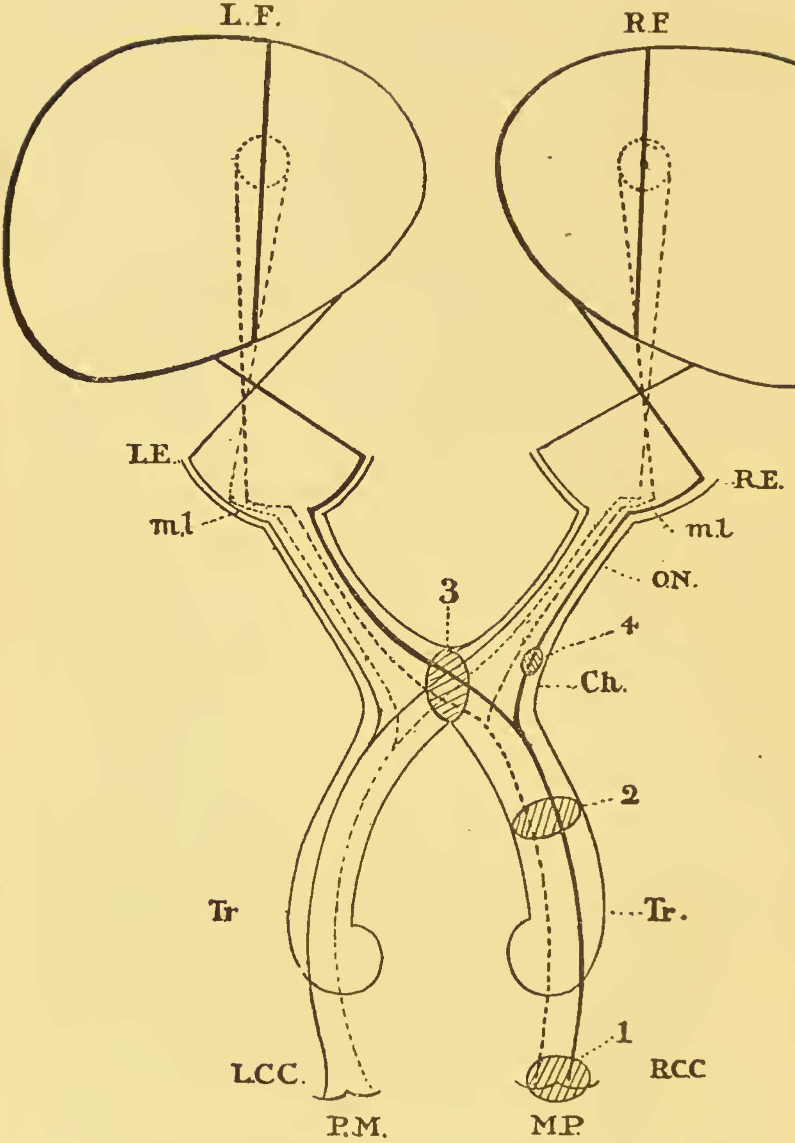

Fig. 10 (Swanzy).—Diagram of course of optic fibres with the cortical centres and relations to fields of vision. R.F., right field of vision; L.F., left field of vision; R.E., right eye (retina); L.E., left eye (retina); O.N., optic nerve; *Ch.*, chiasma; *Tr.*, optic tract, ending in a bulbous enlargement—the

optic thalamus, corpus geniculatum, and corpus quadrigeminum; L.C.C. and R.C.C., left and right cortical centres in the occipital lobe. Between the bulbous enlargement and R.C.C. are the right optic radiations (here made too short); P.P., ending of fibres in right and left cortical visual centre. The fibres coming from the centre of vision to the macula lutea (*m.l.*) are here represented as having the same arrangement as those from the periphery; M.M., ending of macular fibres in visual cortical centres. 1. Lesion of right cortical centre = left homonymous hemianopia. 2. Lesion of the right optic tract = left hemianopia. 3. Lesion of the chiasma = bi-temporal hemianopia. 4. Lesion involving lateral bundle only to right eye, causing nasal hemianopia in the right field. Compare figs. 18 and 19.

of light which reach each eye on its inner side and impinge on the outer half of each retina. The field of each eye is divided into a right field and a left field by the optic axis—a line drawn forwards to the fixation point from the central point of the retina.

In each hemisphere is represented the corresponding half—right or left—of the field of vision of each eye, and for the purpose of illustration we will take the left half of the field of either eye. As seen in the diagram (fig. 10), the left field of the left eye is, owing to the action of the lens, focussed on to the right (nasal) half of the left retina, and the left field of the right eye on to the right (temporal) half of the right retina. The fibres from the right half of the left retina cross over from the optic nerve by the chiasma to the right optic tract, and the fibres from the right half of the right retina are continued uncrossed into the right tract. This tract winds round the under and outer surface of the crus cerebri to the posterior part of the optic thalamus, where it divides into two branches. The larger branch goes to the corpus geniculatum externum, the posterior part or pulvinar of the optic thalamus, and the anterior corpus quadrigeminum, and these ganglia are connected with vision as they are found to degenerate when the eye is extirpated in young animals. From these three bodies fibres pass backwards lying posteriorly to the internal capsule, and course outside the posterior part or cornu of the lateral ventricle, forming what is known as the optic radiations, to end in the cuneus and lingualis, the two convolutions above and below the calcarine fissure on the median surface of the right occipital lobe.

From this it will be seen that destruction of the right occipital convolutions, of the right optic radiations, of the three grey ganglia on the right side with which they are connected, or of the right optic tract, will produce loss of vision for objects to the left of the middle line of both eyes or left homonymous hemianopia. Similarly lesions of these parts of the brain on the left side will cause loss of vision for objects to the right of the middle line of each eye. Therefore a lesion of the right hemisphere would cause paralysis of the left arm, and also loss of vision of the left half of the visual field, where the left arm habitually works.

Besides the occipital cortex, the angular gyrus has been considered as probably a higher visual centre in which both optic tracts are represented, so that a lesion there would cause great impairment of vision of the whole of the opposite eye with much concentric limitation of the peripheral field, and also to a less degree of the eye of the same side.

The other or smaller root of the optic tract goes to the corpus geniculatum internum, and to the posterior corpus quadrigeminum, and perhaps has to do with the relation of the ocular movements to vision and to the reflex contraction of the pupil to light.

The path of the **auditory fibres** is not so well known as that of the optic. The auditory part of the eighth nerve comes from the cochlea, and on reaching the medulla crosses to the opposite side, and then ascends probably by the tract called the lateral fillet or lemniscus to the posterior corpus quadrigeminum and the corpus geniculatum internum; thence fibres pass through the internal capsule behind the sensory fibres to the first temporo-sphenoidal convolution—the auditory centre.

Having now described the anatomy of the chief parts of the nervous system, we shall be in a position to trace the paths of the different sensory and motor impulses according to the scheme set forth in Chapter I.

Reflex action.—As stated in Chapter I., the cord has in its grey matter centres for reflex action; the chief being the cutaneous, the vesical, sexual, and anal, and in the medulla those for vomiting, coughing, and sneezing. The simplest form is the cutaneous, such as the plantar, which may be abolished by any lesion which affects the sensory afferent nerves, the posterior or anterior horn in the cord, or the motor nerves and the muscles they supply. It is through a sensory stimulus such as tickling the sole of the foot, which is carried up to the cord by the posterior roots and thence by fibres which are sent forwards and form a plexus round the cells of the anterior horns, that a motor impulse is started in these cells, which travels down to the muscles by the motor root (see fig. 13). In the anal and vesical reflexes the mechanism is similar to the skin reflexes; the sensory impressions arise from the distension of the rectum or bladder, and are carried up to the lumbar enlargement of the cord, where they give rise to the motor impulses. In the case of the rectum the following arrangement exists. The contents are prevented from escaping by the continued tonic action of the sphincter ani; when, however, a sensory impression is carried up to the cord and acts on the motor part of the centre, the sphincter is relaxed, and at the same time the muscular wall of the rectum contracts and expels the contents. The vesical reflex is similar, and the motor part probably consists of two different centres, one which keeps the sphincter in a continued state of tonic contraction, and the other which calls into action the muscular fibres of the bladder. When the bladder is full, the sensory impression carried up to the cord relaxes the tonic action of the sphincter centre and stimulates the detrusor centre for the contraction of the bladder, and the contents are discharged.

The cutaneous, anal, and vesical reflexes are connected with the cerebrum by afferent and efferent fibres, so that the sensory impressions carried to the cord are appreciated in the cerebrum, and then the motor action of the reflex centre in the spinal cord can be prevented from acting, or in other words a person in health feels when the bladder is full, and yet is able for a time to prevent it being evacuated.

"**Tendon reflexes.**"—These so-called reflexes are obtained by stretching a muscle, either by striking its tendon, or by directly extending it. This is followed in health by a single contraction of the muscle, and is best illustrated by the knee jerk, the contraction of the extensor cruris on striking the patellar tendon. The vastus internus appears to be the part of the extensor which is essential for the knee jerk (Sherrington). Although the time taken by the phenomenon is too short for a true reflex action (*i.e.* to travel up to the cord and back again), the tone of the muscle is so altered by the division of either the sensory or motor root (fifth lumbar, according to Sherrington, for the knee jerk), or by the destruction of the grey matter of the cord, that the reaction is then absent. Hence the knee jerk is lost in locomotor ataxia from disease of the posterior roots, in infantile paralysis from disease of the anterior horns of the cord, in neuritis from disease of the peripheral nerves, and in pseudo-hypertrophic paralysis from disease of the muscle.

Increase of these so-called tendon reflexes is produced by sclerosis of the lateral columns of the cord, so that the slightest blow on the patellar tendon produces a vigorous contraction of the extensor cruris. Ankle clonus is a series of rhythmical contractions of the calf muscles produced by stretching the tendo Achillis by forcibly dorso-flexing the ankle; it is absent normally, but produced in cases of lateral sclerosis.

Sensory impressions from the skin and muscles enter the cord by the posterior root (figs. 11, 13), whose fibres divide into an ascending and a descending branch in the postero-external column or in the posterior horn, whence they probably ascend by the posterior median column and by the opposite antero-lateral tract, either directly or with the intervention of a posterior horn cell, passing across to it by the anterior commissure. Some fibres go directly forwards as branching collaterals to the neighbourhood of the anterior and the posterior horn cells, and are probably concerned with reflex actions. The path of the sensory fibres is still very uncertain, and at present all we can say is that, when one-half of the cord is cut across, although the cord degenerates upwards in the posterior median and

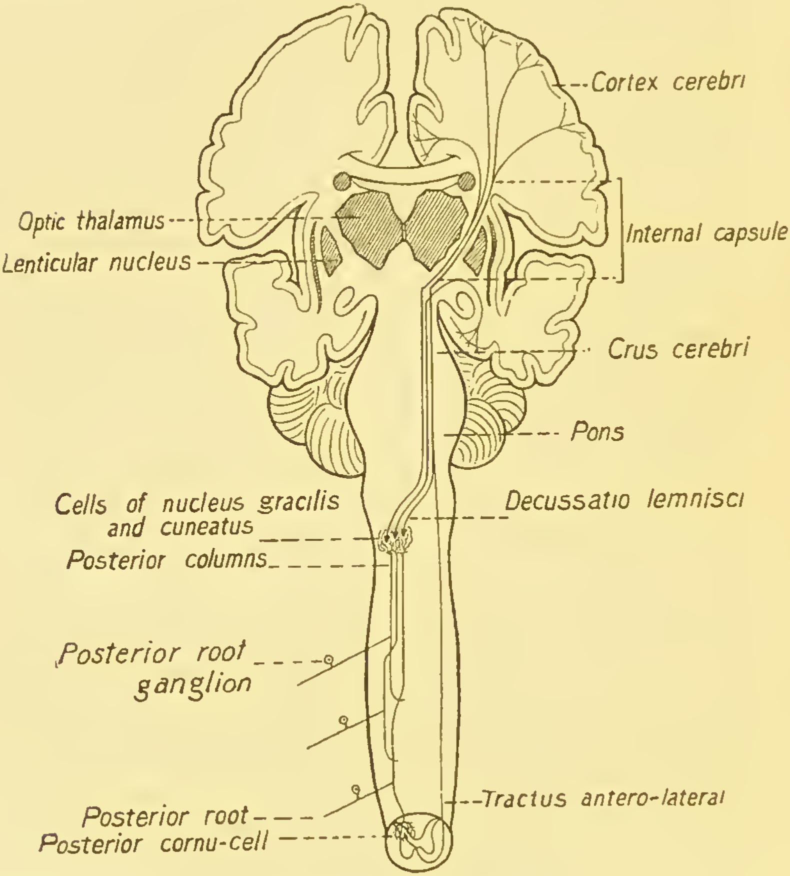

Fig. 11.—Diagram of path of sensory fibres from the periphery to the cortex.

postero-external columns of the same side (as well as in the direct cerebellar and antero-lateral tracts lying along the periphery of the cord), in man this lesion is usually followed by loss of sensation on the opposite side. Probably tactile and muscle impressions are conveyed by the posterior median column, while painful and temperature impressions are conveyed by the antero-lateral tracts, or by the central grey matter.

It must be stated that, according to the experimental work of Mott, hemisection of the cord in monkeys produces loss of power and loss of sensation on the same side. In this case these sensory fibres would ascend by the posterior columns of the same side, and would cross to the opposite side by what is known as the decussation of the lemniscus (fillet), situated just above the decussation of the pyramids in the medulla.

Rather less is known of the course of the sensory fibres in the medulla and pons, but it is situated probably in the posterior part known as the lemniscus, which receives the internal arcuate fibres from the posterior columns crossing through the decussation of the lemniscus, and also the fibres from the antero-lateral tract of the cord, which have crossed over below.

Sensory impressions are probably conveyed by the lemniscus or fillet—which in the crus cerebri joins the part known as the crusta—to the point where the crus cerebri joins the basal ganglia and where the crusta ends in, and is continued on by, the internal capsule. The sensory fibres are contained in the posterior third of the posterior limb of the internal capsule, and pass from this along the corona radiata to the cortex of the parietal lobe, which is considered by some to be the seat of localisation for sensation. Whether this or the gyrus fornicatus be the area where sensations are localised, it seems probable that branches from the fibres of the corona radiata are sent forwards to form a plexus around the cells of the excitable (motor) cortex.

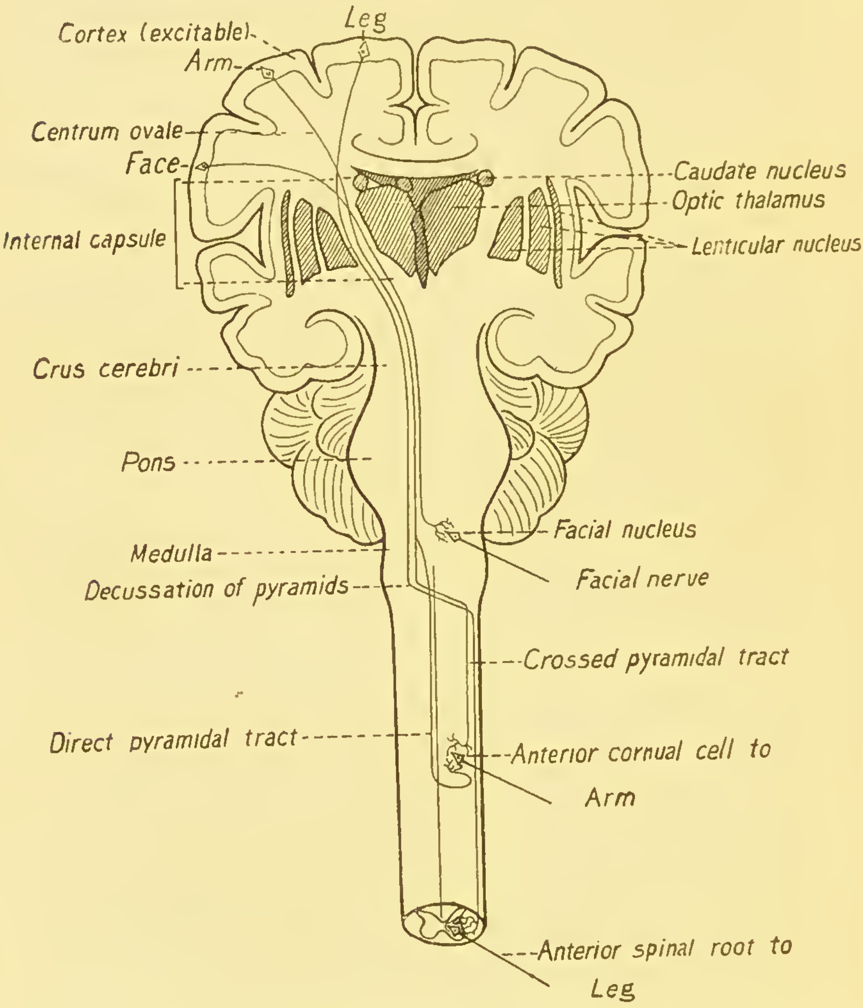

Fig. 112.—Diagram of path of motor tract from the cortex to the anterior spinal roots.

I have also described fibres from the gyrus fornicatus passing in the direction of the excitable cortex.

The motor cortex probably receives fibres from the parts of the cortex where the special senses of sight, hearing, taste, and smell are localised. The most important point clinically is that a lesion of the hinder part of the internal capsule produces opposite hemi-anæsthesia.

Motor excitations.—The large cells of the excitable, or so-called motor, cortex on either side of the Rolandic fissure send down their axis-cylinder processes as fibres through the corona radiata to the excitable part of the internal capsule, occupying the posterior part of the anterior limb and the anterior two-thirds of the posterior limb, then along the crusta of the crus cerebri into the pyramidal tract in the pons and medulla, where fibres are sent to the nuclei of the cranial motor nerves mainly of the opposite side (figs. 12 and 4). The tract decussates with its fellow into the lateral column of the opposite side forming the crossed pyramidal tract, but a small part is continued uncrossed into the anterior medial column, as the direct pyramidal tract along the inner side of the anterior median fissure of the cord.

The lateral columns, as already stated, send fibres into the anterior horns which come into relation with the cells there along the whole length of the spinal cord. The axis cylinders of these cells pass out by the anterior root to end in the muscles.

From the above description it will be seen that on the motor side a lesion may be described as occurring in the following places :—

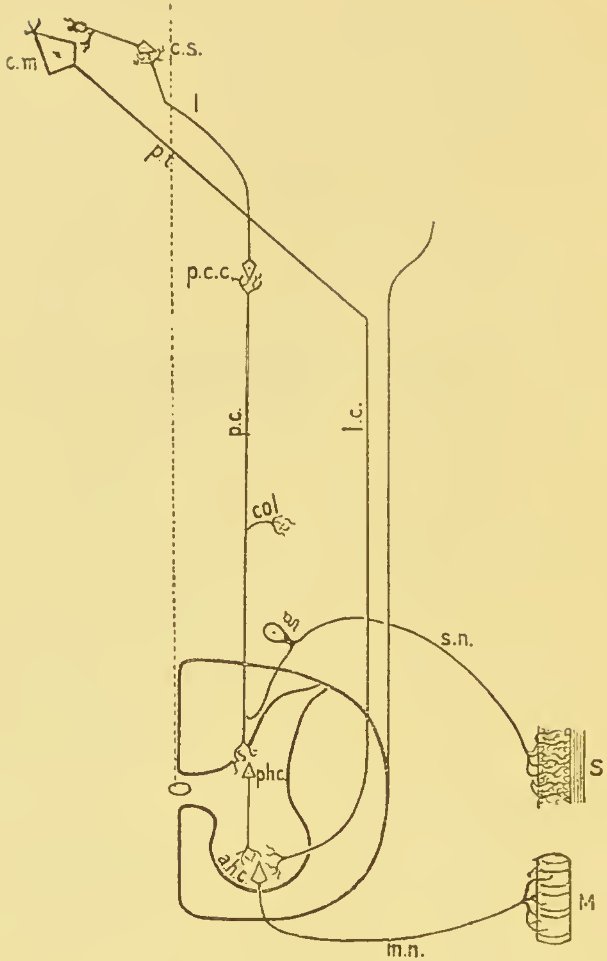

Fig. 13.—Diagram to explain reflex action and secondary degeneration. s., skin with peripheral endings of the sensory nerve, *s.n.*, which passes by the posterior root into the left posterior external column of the spinal cord just beyond *g.*, the ganglion cell on the posterior root. Within the cord one branch is sent forwards into the grey matter forming a network round a cell of the posterior column, *p.h.c.*, which again sends a process forwards around the anterior horn cell, *a.h.c.*, whose axis-cylinder process passes out as the motor nerve, *m.n.*, to the muscle, M. These form the reflex arc.

p.c., the continuation upwards in the postero-median column of the other branch of the sensory root, giving off *col.*, a collateral branch to another posterior horn cell, higher up the cord, and ending in a network round *p.c.c.*, the posterior column cell, the axis-cylinder process of which crosses to the opposite (right) side by the internal arcuate fibres through the decussation of the lemniscus, or fillet, *l.*, and thence gets to the (right) sensory cortex forming a network round the sensory cell, *c.s.*, whose axis-cylinder process forms a network round *c.m.*, the motor cortical cell. The axis-cylinder process from this passes down *p.t.*, the pyramidal tract, crossing to the left lateral column, *l.c.*, to end as a network round the anterior horn cell.

1. Cortex cerebri (so-called motor area).
2. (*a*) Corona radiata.
   (*b*) Internal capsule.
   (*c*) Crus cerebri and pyramidal tract.
   (*d*) Crossed pyramidal tract and lateral column of cord.
3. (*a*) Motor nuclei of pons and medulla.
   (*b*) Anterior horns (and anterior roots) of the spinal cord.
4. Peripheral nerves.
5. Muscles.

Secondary degeneration.—In the motor system, when a lesion affects the cells of the grey matter which forms the starting-point of a motor impulse or the system of fibres connecting this grey matter with the next station of grey matter, all the fibres degenerate from below the lesion as far as the next station. For instance, a lesion of a motor cell of the cortex (*c.m.*) (fig. 13) will cause degeneration of the whole length of its axis-cylinder process as far as, but not affecting, the anterior horn cell (*a.h.c.*), the next station below it;

and the effect will be the same from a lesion in any part of this axis-cylinder process in the pyramidal tract or lateral column—it will degenerate down from the point of lesion to as far as *a.h.c.* In like manner a lesion of the anterior horn cell (*a.h.c.*) will cause degeneration of the motor nerve and muscle (*m.n.* and M.), and, as we shall see later, wasting of the muscle and electrical changes in the nerve and muscle. Hence in a lesion of the cortex the degeneration extends down the corona radiata, internal capsule, crus cerebri, pyramidal tract, the direct tract, and the crossed pyramidal tract in the opposite lateral column of the cord.

Similarly a lesion of the cell (*g.*) of the ganglion on the posterior root will cause degeneration of its prolongation along the sensory nerve (*s.n.*) to the skin, and also upwards along its continuation up the posterior columns of the cord (*p.c.*) as far as the network round the cell of the posterior column (*p.c.c.*), and a lesion in this cell will cause an ascending degeneration as far as, but not affecting, the sensory cell (*c.s.*) of the cerebral cortex.

The degeneration depends on the Wallerian law—*viz.* that separation of a nerve fibre from the nerve cell, with which it is connected and from which it has grown, is followed by degenerative changes; and this law has increased significance from recent investigations in the relation of the fibres to the cells (Golgi, Ramón y Cayal). As is shown by the diagram (fig. 13), the large cell of the motor area of the cortex sends down its axis-cylinder process, which extends down the corona radiata, crus cerebri, pyramidal tract, to the cell of a motor cranial nerve (as the facial), or to the cell of the anterior horn in the spinal cord (as for a muscle of the thumb); according to the above investigators, it does not join this cell, but breaks up into a network, or arborisation, which surrounds the cell. In like manner this cell of the cranial nerve nucleus or of the anterior horn sends out its axis-cylinder process along the anterior root, and it is continued down to the muscle. Consequently, when either cell is destroyed or damaged, its axis-cylinder process degenerates down to its ending in the arborisation in the one case and in the muscle in the other. The protoplasmic processes of the nerve cells are considered to be for nutritional purposes, in connection with the perivascular lymph spaces, and do not join with the processes of other cells. The axis-cylinder process sends off collateral branches, which in the case of the cortical cells may pass across the corpus callosum and end in arborisations round the cortical cells there.

Cerebellum.—This part of the nervous system occupies the posterior fossa of the cranium, and consists of two hemispheres joined together by a middle lobe, the grey cortex of which is composed of leaves or lamellæ, and contains the large cells of Purkinje arrayed in a single layer. Within the cortex is the central white matter.

Each hemisphere has three peduncles—superior, middle, and inferior. The inferior peduncle is the continuation of the restiform body, which receives the ascending cerebellar tract, and perhaps others from the spinal cord, and ends in the white matter of the hemisphere. The middle peduncle is connected with the central white matter, and passes out horizontally into the pons, where the fibres cross to the opposite side, and are connected with the cells of the grey matter which is found in the anterior part of the pons. The superior peduncle starts from the dentate nucleus, a mass of grey matter within the central white matter, and passes upwards, forming the upper posterior boundary of the fourth ventricle under the corpora quadrigemina; it then decussates with its fellow, and either ends in the optic thalamus or in the cerebral cortex.

Physiologically the cerebellum is the important organ in regulating balance, and in co-ordinating movements which preserve the equilibrium. Therefore, from a clinical point of view, the lesions of the cerebellum are recognised by the absence of definite paralysis, or loss of power of movements, and the presence of inco-ordination, especially on standing or walking, giving rise to the cerebellar reel. According to Nothnagel there is no inco-ordination unless the middle lobe is involved; according to Ferrier and Turner, removal of one lateral lobe produces tremors and inco-ordination of the limbs of the side of the lesion, while, according to Risien Russell, it produces in addition reeling to the opposite side, rotation round the longitudinal axis so that the face turns to the side of the lesion, and also slight paresis, rigidity, anæsthesia, and increased knee jerk, all on the side of lesion.

Therefore, according to Russell, a lesion of the *left* lateral lobe would cause staggering and reeling to the *right*, and rotation with the face to the left, or, as I should like to describe these cases, rotation in the direction of "unscrewing a corkscrew," where the handle of the screw corresponds to the animal's head, but inco-ordination, slight weakness, slight anæsthesia, with rigidity and increased knee jerk in the *left* limbs, and in addition inco-ordination of the eyes, as shown by lateral nystagmus to the *left*. Of the peduncles, section of the middle one on the left side causes the animal to rotate round his longitudinal axis in a direction of from left to right when viewed from the front (Ferrier), or in the direction of screwing in a corkscrew.

Vessels of the brain and spinal cord.—The arterial supply for the brain comes from two sources—the internal carotids and the vertebrals. The left common carotid springs directly from the highest part of the arch of the aorta, the right comes from the innominate. The vertebrals unite to form the basilar artery which runs along the front of the pons, to which it gives branches, and between the crura cerebri it divides into the *posterior cerebrals*, which turn outwards and backwards.

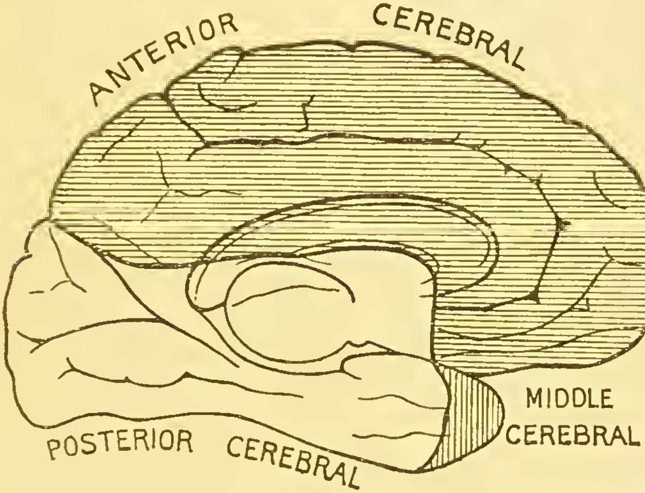

Fig. 14.—Diagram of the arterial distribution on the median surface of the left cerebral hemisphere. The area supplied by the anterior cerebral is shaded horizontally, that by the middle cerebral is shaded vertically, while the unshaded part is supplied by the posterior cerebral.

The *internal carotid* is continued into the *middle cerebral*, and as it ends it gives off the *anterior cerebral*, which runs forwards and courses over the corpus callosum between the hemispheres. Just in front of the optic commissure the arteries of the two sides are joined by the anterior communicating artery, and from each internal carotid a posterior communicating branch is given off to the posterior cerebral. This arrangement is known as the circle of Willis.

The distribution of the middle cerebral artery is the most important clinically; it passes outwards along the fissure of Sylvius to the outer surface of the hemisphere, where its branches reach in the parietal region to the middle line. Its distribution is twofold: (1) to the interior of the hemisphere; (2) to the outer surface.

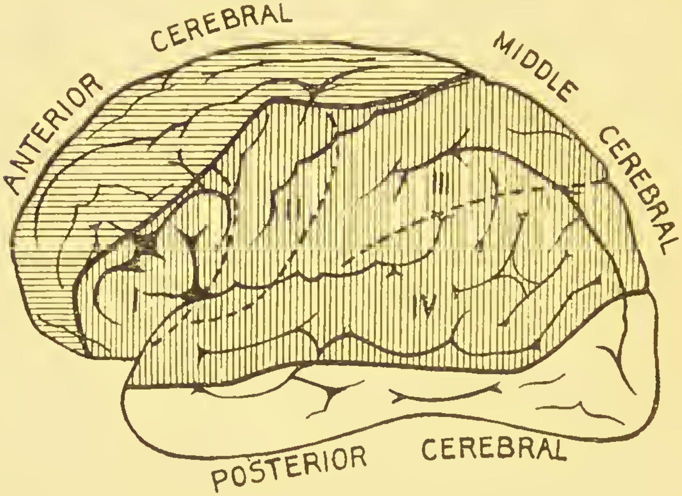

Fig. 15.—Diagram of the outer surface of left hemisphere showing the arterial distribution. The shading as in fig. 14. I, II, III, IV, the areas supplied by the four branches of the middle cerebral.

(1) Several branches are given off from the first inch of the course of the artery, which pass into the anterior

perforated spot (just in front of the optic tract). These are distinguished by Duret into internal, which are small, and pass upwards to supply the inner part of the lenticular nucleus and the median part of the internal capsule; and external, which course along the base of the lenticular nucleus and turn upwards into its outer part and through the internal capsule. Some of these external arteries run forwards and upwards, to end in the caudate nucleus of the corpus striatum, and are called lenticulo-striate; and others run backwards, to end in the optic thalamus, and are called lenticulo-optic —these are very liable to hæmorrhage. The choroid arteries come off from the middle cerebral, and pass backwards to enter the lateral ventricle.

(2) On reaching the outer surface of the hemisphere (fig. 15) the middle cerebral divides into four branches, which supply (i) the third frontal convolution ; (ii) the ascending frontal (lower two-thirds) and second frontal (posterior part); (iii) the ascending parietal and the parietal lobule ; (iv) the supra-marginal, the angular, the first two temporo-sphenoidal convolutions (posterior part), and the first two temporal convolutions (anterior part), as well as the hippocampal lobule on the median surface.

The anterior cerebral (fig. 15) supplies the rest of the outer cortex in front of the middle cerebral distribution, and also all the median cortical surface (fig. 14) as far back as the internal parieto-occipital fissure, and in front the supra-orbital lobule of the base of the brain ; at the commencement a few branches are given off to the head of the caudate nucleus.

The posterior cerebral artery, after winding round the crus cerebri, gives branches to the posterior part of the optic thalamus, the crus cerebri, the corpora quadrigemina, and the posterior part of the internal capsule ; it then supplies the outer surface (fig. 15) below the middle cerebral distribution, and all the median surface of the hemisphere (fig. 14) which is not supplied by the anterior cerebral. The branches from the different arteries anastomose to a certain amount; but this varies much in different individuals. The cerebellum is supplied by (1) the superior cerebellar from the basilar, (2) the middle cerebellar from the basilar, and (3) the posterior cerebellar from the vertebral.

The venous circulation of the cortex is peculiar, as the blood goes the same way as in the arteries. On the convexity of the hemispheres the blood enters the superior longitudinal sinus forwards against the blood stream, and as there are no valves in the cerebral veins or sinuses it explains the facility with which thrombosis occurs. The superior longitudinal sinus ends posteriorly in the torcular Herophili. At the base of the brain the cavernous sinus receives the veins from the anterior part, the petrosal sinuses from the middle part, and the lateral sinuses from the posterior part of the brain; the veins corresponding to the choroid plexus in the lateral ventricles, returning blood from the interior of the cerebrum, end in the veins of Galen, which discharge through the straight sinus to the torcular Herophili and thence through the lateral sinus. Blocking of the veins of Galen gives rise to distension of the ventricles by fluid. Nearly all the blood leaves the cranial cavity by the internal jugular vein, the continuation of the lateral sinus, the cavernous sinus draining through the petrosal to the lateral sinus; the other most important channels are the connection of the veins of the nose with the superior longitudinal sinus, the ophthalmic with the facial vein, and the lateral sinus through the mastoid cells with the occipital vein —a condition associated with mastoid disease and thrombosis of the lateral sinus.

The arterial supply of the cord is derived from one anterior spinal artery running the whole length of the cord and sending branches into the anterior median fissure, which bifurcate at the anterior commissure and supply the anterior horns and the base of the posterior horns; and two posterior spinal arteries, which give off small branches extending from the posterior median fissure to the anterior roots, supplying the white matter. The spinal arteries receive their blood from the spinal branches of the intercostal and lumbar arteries, which enter each intervertebral foramen. The venous blood is returned by anterior and posterior spinal veins, which discharge by veins through the intervertebral foramina.

CHAPTER III.

METHOD OF TAKING A CASE.

In nervous diseases, perhaps more than in any other branch of medicine, it is of the greatest importance to examine the patient in a systematic manner, and to follow some plan which can easily be remembered.

In the family history, particular inquiry should be made of any nervous disease or insanity, as well as of malignant disease, tuberculosis, and gout.

In the previous history, syphilis, rheumatic fever, gout, and Bright's disease, as well as any previous nervous disease, should be especially noted.

In the history of the case, the mode of onset, whether sudden or gradual, is of great importance; and it is also very necessary that the facts should be extracted from the patient or his friends without giving leading questions.

In describing the *present state*, there are two different plans which may be followed: (1) the regional or anatomical method, where a region such as the head or face is taken and everything described there, including motion, sensation, the organs of special sense, etc.; (2) the physiological method, where each system—*i.e.* motor, sensory, etc.—is taken separately and described throughout.

There is little doubt that the latter is the more scientific, and that by following it there is much less likelihood of leaving any part unexamined, and it is also very much easier to find what we want in notes taken this way. The exception to this method is in the case of where several cranial nerves are involved, when it is useful to examine each separately and tabulate the result.

After describing the general state and especially the expression of the features and the general attitude of the patient, the mental condition must be noted, whether comatose, delirious, obtuse, memory deficient, or suffering from delusions.

We should then examine :—

The motor system.—Noting (1) any wasting of the muscles, (2) rigidity or spasm, (3) tremors, (4) any loss or diminution of voluntary movement, (5) inco-ordination, (6) convulsions. We should test specially the—

Movements of the *eye-balls* in all directions, convergence, nystagmus, strabismus, diplopia. *Pupils*—size, reaction to light, direct and lateral, and to accommodation.

Face—closing eyes, elevating and wrinkling brow, elevation and retraction of angles of mouth, inflating cheeks.

Mouth—closing and opening, grinding teeth, elevation of soft palate, protrusion and other movements of tongue.

Throat—swallowing, movements of vocal cords.

Speech (see " Defects of speech."

Head—flexion and extension, rotation and lateral movement.

Upper limb.—*Hand—thumb*, flexion, extension, abduction, adduction and opposing; *fingers*, flex and extend, separate and adduct. Buttoning clothes, writing.

Wrist—flex and extend, abduct, adduct; pronation and supination of forearm.

Elbow—flex and extend.

Shoulder—advance, retract; abduct, adduct; rotate out and in.

Scapula—advance and retract, elevate (shrugging shoulders).

Spine—curvature, lateral or angular, lordosis, cyphosis; tenderness to percussion; flexion, extension, lateral movements, rotation.

Respiration—thoracic, diaphragmatic.

Abdominal muscles—movements of trunk forwards, coughing.

Lower limb.—*Hip*—flexion and extension, rotation in and out, abduction, adduction.

Knee—flexion and extension, rotation (after flexion).

Ankle—dorso-flexion, plantar extension, eversion and inversion.

Toes—extension and flexion.

Standing, walking, getting up from the prone to the standing position.

Electrical reactions of the different muscles to (1) faradism, (2) the constant current.

Reflexes.—(1) *Deep*—knee jerk—present, absent, or excessive, rectus clonus.

Ankle clonus—present or absent.

Wrist tap—present or excessive.

Lower-jaw jerk—present or as clonus.

(2) *Superficial*—epigastric, abdominal, cremasteric, plantar; soft palate, conjunctiva.

Organic reflexes.—(1) *Vesical:* (i) Incontinence—(*a*) simple—urine runs away as fast as it enters bladder; (*b*) intermittent—the urine is expelled automatically; (*c*) overflow incontinence with retention. (ii) Complete retention.

(2) *Anal:* incontinence of fæces—(i) from paralysis and relaxation of sphincter; (ii) from removal of voluntary influence.

Trophic lesions—bullæ on skin, bed sores, perforating ulcers of foot, changes in colour and temperature of limbs, and joint changes.

Temperature of the two sides of the body.

Sensory system.—Character of pains in the head, limbs, and trunk; tenderness on pressure over the chief nerve trunks, muscles, and skin. Tingling, pins and needles. Tenderness of head and spine to precussion or pressure.

Loss of sensation,—to (1) tactile, (2) painful, (3) temperature, impressions over the whole surface of the body, including the mucous membrane of the eyes, nose, and mouth. (4) Muscular sense—(*a*) Loss of appreciation of passive movements and position of all the joints, (*b*) appreciation of different weights, (*c*) exact localisation of the place touched.

Delay of appreciation of sensations.

Organs of special sense.—*Sight,* acuity, field of vision, hemianopia, peripheral limitation; colour vision. Subjective sensations—flashes of light, colours, "zigzags," photophobia. Optic neuritis ; optic atrophy, grey and white.

Hearing—aerial conduction ; perosseal. Subjective noises in the ears, tinnitus aurium.

Taste for sweet, bitter, acid, and salt sensations in (1) the tip and edges of tongue, (2) the posterior part of tongue and the palate, on each side.

Smell for odorous substances, as volatile oils, assafœtida for each nostril.

Examination of other organs, especially the circulatory and the renal systems.

In examining the mental condition of a patient, we should ascertain if he is comatose, and the degree of insensibility ; whether the pupils react to light ; if the conjunctival reflex is present ; if the breathing is natural, or stertorous with blowing out of the cheeks ; whether he can be roused by shouting ; whether delirious, or merely stupid and obtuse.

The subsequent examination will depend very much on the amount of assistance given by the patient, which, if the mental condition is much affected, may be *nil*, and then we have to rely on what we can observe for ourselves.

General diagnosis.—After taking accurate notes of the present state of a case, the next point is to make a diagnosis ; and here it will be advisable to say a few words as to the mode of sifting the evidence, and determining in which part of the nervous system the disease has its lesion.

We may divide the diseases of the nervous system into—(1) organic diseases ; (2) general and functional diseases. In the first group are included diseases of peripheral nerves, of the spinal cord, of the pons and medulla, and of the brain, in which pathological changes are found after death. In the general and functional group are included such diseases as chorea, paralysis agitans, epilepsy, neuralgia, hysteria, and neurasthenia, diseases in which the pathological changes are unknown or doubtful.

In the first group the diagnosis is arrived at by the distribution, grouping, and extent of the paralysis of the muscles, and by the presence or absence of wasting with or without electrical changes; the extent and area of the loss of sensation; the condition of the sphincters; the mental condition, such as loss of consciousness, delirium, headache; the absence or presence of optic neuritis; and by the condition of the deep reflexes.

The extent, distribution, and grouping of the paralysed muscles will correspond to the anatomical motor supply of a peripheral nerve, when the ulnar or median nerve is affected, and the loss of sensation will correspond to its anatomical sensory distribution. In multiple neuritis many of the peripheral nerves are involved symmetrically, and parts of all four limbs suffer. In spinal-cord diseases the distribution is usually bilateral, causing paraplegia, or if the lesion is high up, paralysis of all four limbs, the face escaping; while in cerebral lesions of one hemisphere the paralysis and the anæsthesia affect the whole of one-half of the body, face, arm, and leg, and is called hemiplegia.

It is very important to note that, whereas in paralysis of a peripheral nerve the distribution is purely anatomical, in cerebral cases excluding cranial nerves it is a paralysis of "movements," and all the muscles taking part in this movement are involved, so that the distribution is physiological.

The rapid or gradual wasting of muscles with loss of electrical reactions is the most certain sign that either the nerve to the muscle, or its nerve cell in the nucleus of a cranial nerve or in the anterior horn is involved, and is of the greatest use in localising the lesion in the motor cells of the pons, medulla, and spinal cord, or the nerves coming from them.

The condition of the deep reflexes follows closely the condition of the muscles. Loss of the knee jerk on one side, especially if associated with wasting and loss of power and of electrical reactions of the muscles, signifies a lesion of the peripheral nerve or of its cells in the anterior horn, and if unattended by anæsthesia probably means the latter; if there is no wasting or loss of electrical reactions in the muscles which subserve the reflex, the lesion is probably on the sensory side of the reflex arc;[1] and if the loss of the knee jerk is on both sides the lesion is probably spinal (*i.e.* locomotor ataxia). Increase of the deep reflexes, if bilateral, is usually of spinal origin (sclerosis of the lateral columns); if unilateral, and affecting the arm and leg, it is probably cerebral, exceptions being unilateral pressure on the cord from caries or a growth, and a lesion in both hemispheres.

The extent, area, and character of the anæsthesia are of great value. The area of anæsthesia in a peripheral nerve lesion corresponds to the anatomical distribution of the nerve; in that of a spinal-cord root it affects the limb in strips running lengthwise, or round the trunk horizontally; in cerebral lesions of one hemisphere it affects the one-half of the patient, the whole of one limb or the lower half of it up to the elbow or knee.

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[1] Exceptions to this are found in the myopathies as pseudo-hypertrophic paralysis, where the knee jerk is lost from primary disease of the extensor cruris muscles.

The condition of the sphincters being affected is greatly in favour of the disease being due to a lesion of the cord, as this does not occur in peripheral neuritis, or in cerebral diseases, except in unusual cases of double hemiplegia or where mental symptoms are present.

Symptoms such as loss of consciousness, delirium, headache, purposeless vomiting, and optic neuritis show that the brain is involved; though optic neuritis usually means cerebral disease, primary optic atrophy is a frequent symptom in locomotor ataxia, the chief lesion of which is in the spinal cord.

To sum up, loss of power in muscles and anæsthesia, having the known anatomical distribution of some peripheral nerve, and attended by wasting, loss of electrical reactions, pain, and loss of deep reflexes, is probably due to a neuritis of this nerve; the symptoms are the same for a lesion of one or more of the spinal roots, except that the grouping of the muscles and the area of anæsthesia are different (see table and diagram at end of book). Atrophy and flaccidity of muscles with paralysis and loss of electrical reactions and of the deep reflexes without anæsthesia mean a lesion of the cells of the anterior horns or of the cranial motor nuclei.

Paralysis of both legs, or of both arms also, with or without anæsthesia up to a certain level on the trunk, with increase of the deep reflexes and affection of the sphincters, means a lesion of the spinal cord probably in the mid-dorsal region, or if the arms are affected in the mid-cervical cord. Paralysis of one-half the patient, including the face, with or without anæsthesia, with increase of the deep reflexes on that side and no affection of the sphincters, is caused by a cerebral lesion or by one in the pons, and if preceded by loss of consciousness the diagnosis is made more certain. Certain parts of the brain, as the frontal lobes and the cerebellum, are not attended by definite paralysis; but as the symptoms of the latter are usually caused by a tumour, the headache, vomiting, optic neuritis taken with the staggering gait, will help the diagnosis.

The general and functional diseases are arrived at after the organic diseases have been excluded; and it is absolutely essential that the latter should be carefully sought for and excluded before a diagnosis of such diseases as hysteria and neurasthenia is arrived at. Many of the general diseases, such as paralysis agitans, can be recognised at once by their symptoms; while in others, as migraine, we depend entirely on the history and the personal account of the patient.

CHAPTER IV.

MODES OF EXAMINATION.

MOTOR SYSTEM.

In the method described in the last chapter, only the chief headings are given of the different parts of the body, and it will be here advisable to explain the way in which the examination is carried out.

We must ascertain, either by observation or measurement—(1) The *size of a limb thought to be wasted*, and compare it with the opposite limb, noting carefully if (*a*) the whole limb is wasted, or (*b*) any definite grouping of muscles; the measurement in the case of the calf or the forearm should be the largest obtainable, and in that of the thigh the identical place—say four or five inches above the patella—should be measured in either limb and recorded.

(2) *Rigidity* is tested by getting the patient to relax the muscles as much as possible, and then passively bending the joint; rigidity due to adhesions in the joints may require an anæsthetic for their demonstration, which will relax the spasm of the muscles, but not the rigidity due to the adhesions or to permanently shortened muscles.

(3) *Tremors* must be noted—(*a*) In the exact part where they occur.

(*b*) Whether rhythmical or irregular in time; if irregular, whether sudden jerking movements (chorea) or slowly changing (athetosis).

(*c*) Whether a repetition of the same movement in the same joint, or different movements in various joints.

(*a*) Whether increased, diminished, or produced only by voluntary effort.

(4) In ascertaining alteration in *voluntary movements*, we must note (*a*) whether any movement is absolutely lost, or (*b*) whether the movements, though performed, are weak. In the first case a patient may have completely lost one movement—*i.e.* extension of the wrist and fingers; in the other, he may perform all the movements of the joint, but with much diminished power, which may be ascertained by an instrument for registering the grasp, called a dynamometer, or by making the patient flex or extend the joint against resistance. It must be particularly noted if the weakness is due to one of the muscles acting on a joint being paralysed, as, for instance, the ulnar flexor of the wrist being affected and not the radial.

(5) *Inco-ordination* of the upper limbs is best tested by making the patient touch his nose with his forefinger with the eyes open and then closed, taking care that he looks at his finger in the first case, and does not steady his arm against his chest in the second; in the lower limbs by making him, when lying down, touch the knee with the opposite heel with the eyes open and shut; also by getting the patient to stand barefooted, with the toes and heels together or a little apart, with the eyes open, and when quite steady, to notice if he staggers more with closed eyes; and by making him walk toe and

heel along a straight line, taking care that the heel as it is put down touches the toes of the other foot.

(6) In *convulsions* it is of the utmost importance to see the commencement, and to notice—(*a*) The part first affected—*e.g.* angle of mouth, thumb, big toe.

(*b*) The nature of the movement—*e.g.* flexion or extension.

(*c*) The character of the convulsion, tonic or clonic.

(*d*) The order in which the other joints are attacked by the spasm—*i.e.* its spread or march.

(*e*) The duration of the fit, as timed by a watch.

(*f*) The condition immediately after the attack—*i.e.* pupils, colour of face, position of eyes, condition of knee jerk, and ankle clonus. In the attack the pupils should be noted, their size and reaction to light, and the conjunctival reflex tried. Tongue biting and involuntary micturition should be looked for.

In convulsions the different parts are involved so quickly that it is quite impossible to remember accurately the order, and the observer should at once dictate notes to an assistant or nurse, who should write them down as they are observed, with, if possible, the time recorded in seconds.

Movements of the eyeballs are tested by fixing the patient's head, and making him follow the finger outwards and up and down, noticing any deficiency of movement, and nystagmus or jerking movements of the eyeballs.

In each eye movement outwards is performed by the external rectus, and inwards by the internal rectus; the movements upwards and downwards are more complicated, and are each performed by two muscles.

As the eyeball not only moves up and down, but also rotates, it is best to take a point on the iris, and notice if it rotates like a wheel round the pupil as its centre. The superior and inferior recti move the eyeball upwards and downwards; but as the origin of these muscles, owing to the axes of the orbits being divergent, is nearer to the middle line of the head than their insertion into the eyeball, the superior rectus will move the eyeball so that the upper edge of the iris goes upwards and inwards, and rotates like a wheel the top of which goes towards the middle line. The inferior rectus will pull the eyeball so that the lower edge of the iris moves downwards and inwards, and rotates the lower edge of the wheel towards the middle line, or the upper edge away from the middle line. To counteract these rotations the inferior oblique muscle works with the superior rectus, and it pulls the eyeball so that the upper edge of the iris moves upwards and outwards, and rotates the upper edge of the iris like a wheel away from the middle line; while the superior oblique, in helping the inferior rectus, draws the eyeball so that the inferior edge of the iris moves downwards and outwards, and rotates the upper edge of the wheel towards the middle line. The oblique muscles are the difficult ones to remember; but by keeping in mind that the inferior oblique works with the superior rectus and *vice versâ*, and then writing down the movements of the superior rectus, which can easily be remembered, it will be found that the inferior oblique agrees in one respect, upward movement, but differs in the other two—*i.e.* the superior rectus moves up, in, and rotates in, the upper edge of iris, while the inferior oblique moves up, out, and rotates out, the upper edge of iris.

Paralysis, with defective movement of one or more of the muscles of one eye, causes strabismus or squint, secondary deviation of the sound eye, and diplopia. In strabismus there is a want of parallelism between the axes of the two eyes, which increases in the direction in which the paralysed muscle acts. Secondary deviation of the sound eye is observed in the following manner in a case of paralysis of the right external rectus. The patient is made to fix an object, placed to the right of the middle line, with the right affected eye, while the left eye is prevented from seeing the object by interposing a sheet of paper along the middle line. It will then be observed that the left eye turns inwards, *i.e.* to the right, more than it normally would; owing to the extra effort which the patient makes to fix the object with the affected eye. This amount of overaction can be measured by removing the sheet of paper, when the left normal eye will be seen to move outwards to fix the object, and this movement gives the amount of deviation.

Diplopia is caused by the object not coming on corresponding parts of the two retinæ, and is tested by fixing the patient's head, and making him follow an object, such as a strip of white paper or a candle, in all directions, when two objects will be seen in the field where the axes are not parallel. The relation of the objects to the eyes is found by putting a red glass over one eye, or by closing one eye and noting which object disappears. Diplopia is said to be simple when the right image is seen with the right eye, and crossed when the right image is seen with the left eye. When the axes of the paralysed eyes, prolonged forwards, would cross (as in internal strabismus), the diplopia is simple; when the axes diverge, the diplopia is crossed. Hence diplopia is crossed in paralysis of the superior, inferior, and internal recti; and simple in that of the superior and inferior obliques and external rectus; and owing to rotation of the eyeball, the false images are inclined to the true image in all except the external and internal recti, where they are erect along the horizontal line. The accompanying diagrams (figs. 16, 17) (L. Werner) enable the observer to remember the position of the false image, whether crossed or not, its inclination, altitude, and the direction of the particular movement required to produce the diplopia, in cases of paralysis of the muscles which rotate the eyeball, where the continuous line shows the true image, and the dotted line the relation of the false image to it, for each separate muscle. So that in paralysis of the right superior rectus the diplopia is crossed, the false image is higher and inclined with its lower end to the true image, and diplopia occurs in upward movement of the eyes.

The *pupils* should be tested singly while in moderate light, by closing the other eye, and then shading the pupil with the hand, and noticing if it dilates to shade, and testing the contraction to light by exposing it to a strong light on removing the hand, and in doubtful cases using a convex lens to focus the light. It is very important that the pupil should be watched all the time, otherwise the patient may converge the eyes, and so produce contraction. Contraction of the pupils to convergence and accommodation is ascertained by making the patient look at his own finger held close

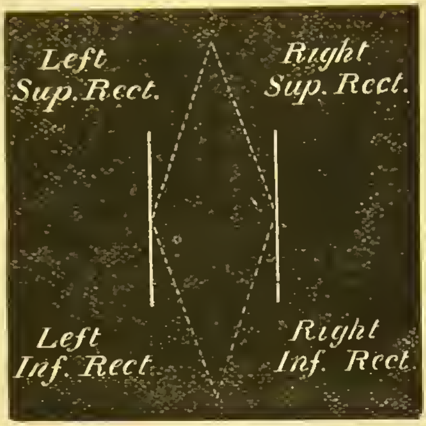

Fig. 16.—Diagram showing position of false image (dotted line) to true image (continuous line), for paralysis of the superior and inferior recti respectively, where the images are crossed (Swanzy, after L. Werner).

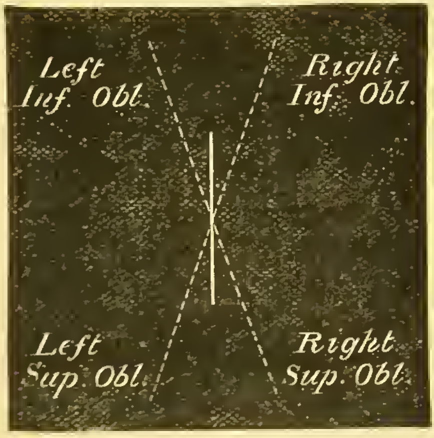

Fig. 17.—Diagram for position of false and true images, for paralysis of the oblique muscles, where the images are not crossed. The true images are considered to be always in the same place, and are combined into one straight line (Swanzy, after L. Werner).

to him, or on the tip of his nose. A pupil which will not contract to direct light will sometimes contract consensually with the pupil of the other eye.

The *orbicularis palpebrarum* is tested by forcibly pushing up the eyelid with the thumb when the eyes are tightly closed.

In examining the movements of the lower jaw to test if the *external pterygoids* are acting equally, the patient should just show the teeth, and the relations of the intervals between the central incisors in both jaws should be noted. If now, on depressing the lower jaw to open the mouth, one condyle is drawn forwards more than the other, the lower jaw will be carried over to the weaker side, and will alter the vertical positions of the spaces between the incisors. The contraction of the *masseters* is readily felt in front of the angles of the lower jaw, when the patient is told to press the teeth together like "cracking a nut."

Paralysis of the *soft palate* can only be accurately ascertained by making the patient phonate, or draw a deep breath, when, if both sides are paralysed, the palate will be seen to hardly move at all, and if only one side is paralysed, the palate is drawn up towards, and puckered on, the opposite side. If both sides are paralysed, the voice has a nasal sound, the patient cannot inflate the cheeks unless the anterior nares are closed with the finger and thumb, and on relaxing this closure, the air escapes through the anterior nares; while regurgitation of fluids through the nose is common in swallowing. Mere hanging of the uvula to one side at rest is no sign of paralysis.

The *tongue* is to be examined for atrophy of one or both sides, when the wasting can be felt by the finger and thumb, and the mucous membrane is thrown into folds, and fibrillar contractions of the muscles are seen. Besides protrusion of the tongue straight or to one side, the movements of the tip upwards and into either cheek should be observed.

In examining the *larynx* with the laryngoscope, the position of the cords and the movement of each should be noted during respiration and phonation. In addition to this, the character of the voice, whether it is absent or changed, the power to cough, and the presence of stridor and impeded breathing, should be observed.

In the following description the complete action of any one muscle is not given, but rather the best position and action requisite to ascertain by palpation and inspection whether a particular muscle is acting—*e.g.* the action of the latissimus dorsi is to put the hand behind the back, but when in that position the muscle cannot be felt or seen, but if the arm be passively abducted, and then the patient be made to strongly adduct the humerus against resistance, the condition of the muscle can be readily ascertained.

In the upper limb, in the *thumb*, flexion and extension take place only in a direction along the plane of the palm and are done by the long muscles of the forearm; abduction, *i.e.* moving the thumb away from the forefinger in a direction at right angles to the palm, adduction, *i.e.* moving the thumb towards the forefinger, and rotation with opposition of the thumb, can only be done by the abductor, adductor, flexor brevis and opponens pollicis, respectively. Abduction is best tested by laying the hand on a table palm upwards and getting the patient to move the thumb upwards at right angles to the palm. The extensor ossis metac. poll. is not an abductor by itself.

The *interossei* are tested by the power of separating and approximating the extended fingers; they also with the lumbricales flex the metacarpo-phalangeal joints of the fingers and extend the phalangeal joints, and when paralysed give rise to the claw-shape hand with hyper-extension of the metacarpo-phalangeal joints and flexion of the phalangeal joints. The *flexores sublimis* and *profundus* flex the proximal and distal phalangeal joints respectively. The fine movements are tested by buttoning.

The *supinator longus*, which is a pure flexor of the elbow, the *biceps* and *brachialis anticus* are tested by making the patient flex the elbow against resistance, when the first stands out prominently along the radial side of the forearm; but care must be taken to fix the humerus in the vertical position, otherwise the patient by rotating the humerus inwards, abducting it and then rotating it out, may allow the elbow to assume the flexed position, when the power of active flexion is completely lost.

The *triceps* is best tested by first fixing the humerus raised vertically above the shoulder, so as to counteract the action of gravity.

The *deltoid* and *supra-spinatus* abduct the humerus to the horizontal line but not further, and the anterior fibres of the deltoid act chiefly when the humerus is advanced (flexed); above the horizontal line the serratus magnus draws the lower end of the scapula, which up to this level has not moved, forwards and the trapezius draws the acromion upwards, and the rotating scapula carries the humerus to the vertical position above the head; in paralysis of the deltoid and supra-spinatus but not of the serratus, the scapula moves forwards at once in attempting to abduct or advance the humerus when it is hanging vertically.

Paralysis of the *serratus magnus* is shown by inability to elevate the humerus beyond the horizontal line, and by the posterior border of the scapula projecting like a wing when the patient advances the humerus to this level, and also by the inability to push horizontally forward with the advanced arm.

In paralysis of the whole *trapezius* the sloping contour of the neck is altered, the scapula is twisted, so that the acromion is rotated downwards, the inferior angle is nearer to the spine, and the whole scapula is rather higher than on the opposite side, being drawn up by the levator anguli scapulæ, which is visible in its whole length. There is much projection of the upper part of the posterior border of the scapula when the humerus is advanced, which diminishes or disappears when the humerus is carried above the horizontal line by the action of the serratus magnus on the lower end of the scapula. Absence of the lower fibres of the trapezius can often only be made out by faradising the muscle, as, if present, fibres running down and in to the spinal column should be seen; also the scapula is a little too high and the inferior angle too near to the spine.

The *rhomboids* draw the scapula backwards and upwards to the spine, and are best tested when the patient stoops forwards rounding the back; when absent the posterior border of scapula is not brought parallel to spine.

The *pectoralis major* as a whole is best brought out on both sides by advancing the humeri, and making the patient press both hands together with the elbows flexed; the upper fibres (clavicular) alone stand out if the patient in this position tries to raise vertically upwards the horizontally advanced arm against resistance, whilst the lower half (sternal) and the *latissimus dorsi* contract strongly on pressing down on a table with the advanced or abducted arm—moreover, when the latissimus is absent, the inferior angle of the scapula in this movement is abnormally drawn forwards by the teres muscles.[1] The strength of the *teres minor* and *infra-spinatus* is shown by rotation out of the shoulder against resistance, and of the *teres major* and *subscapularis* by rotation in (remembering that the pectoralis major is a strong rotator in).

In the chest, respiratory movements may be purely thoracic with paralysis of the *diaphragm*, when the descent of the liver cannot be felt on deep inspiration, and the epigastrium is depressed instead of being protruded; in paralysis of the *thoracic* muscles from a lesion of the cord in the cervical enlargement, the diaphragm alone acts, there is no elevation and expansion of the chest wall, but the lower ribs to which the diaphragm is attached are slightly elevated and rotated outwards.

The *rectus abdominis* of either side can be felt to contract by making the patient attempt to get into the sitting position while lying down without using his arms, being careful at the same time to fix the thighs. If the lower halves of the recti alone are paralysed, the umbilicus is drawn upwards in making this movement.

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[1] This drawing forward of the scapula has been ascribed to want of fixation by the trapezius and rhomboids; in my opinion it only occurs when the latissimus dorsi is absent.

The *erector spinæ* muscles are tested by fixing the pelvis in the sitting posture, and making the patient extend the spine against resistance.

The *gluteus maximus* extends the hip, and is brought into action in swinging the rigid body backwards in the sitting position, as in rowing.

The flexors of the hip, *psoas* and *iliacus*, cannot be felt, but any weakness is ascertained by making the patient flex the thigh against resistance, or try to walk upstairs, as patients can often walk on the level with paralysis of these muscles. It must be remembered that patients with so much paralysis of the *extensor quadriceps* of the knee as to prevent them extending the knee when sitting down, can yet manage to walk by swinging the leg forward and putting the foot down while the leg is straight, as in standing and walking the quadriceps is very little used.

The character of the *walk* is most important, and especially as to whether it is due (*a*) to loss of power of certain movements, (*b*) to rigidity, (*c*) to inco-ordination or a difficulty in carrying out the movements.

Dragging the foot along the ground or circumduction of the leg usually means weakness of the anterior tibial group of muscles as well as of the other muscles of the limb, as in hemiplegia; but if the anterior tibial muscles are alone affected, the walk is high stepping, the knee and hip being overflexed to clear the ground of the hanging foot as the leg is brought forward. If however the extensor cruris and the flexors of the hip are weak, the necessary elevation of the foot is performed by tilting up the pelvis with each step, giving a swaying movement to the body. In rigidity, dragging the foot, circumduction, and swaying may all be present.

The general character of the walk must be noted apart from weakness of particular movements, and especially if there is—(1) Inco-ordination with throwing of the legs about, turning up the toes so as to bring the heels down with a stamp, and unsteadiness in turning round (as in tabes). (2) A tendency to fall over to one side like a drunken person, the amplitude of the movement being much larger than in the previous case (as in cerebellar disease). (3) A gliding walk, either with long steps, or with short quick steps, with a fixed attitude of the body (as in paralysis agitans). (4) General coarse tremor of the leg with each step (as in disseminated sclerosis).

For the *electrical testing*[1] of muscles a combined faradic and galvanic battery should be used, the latter consisting of thirty to forty cells, and fitted with a galvanometer graduated in milliamperes and a current reverser. The secondary current from the faradic should be used. The first essential in testing is, that the skin of the part and the electrodes should be thoroughly wetted by hot salt and water. One pole, the positive, is to be connected with a large flat well-padded electrode four inches long, which is kept in close contact with an indifferent point, as the sternum; and the other, the negative, is attached to an electrode about an

[1] For more detailed accounts, De Watteville's or Lewis Jones's *Medical Electricity* should be consulted.

inch in diameter. In using the currents it is always well, as a rough standard, to try the strength on the small thumb muscles of one's own hand first. In every muscle there is a point which gives when stimulated the maximum effect to the current; this is called the motor point. Charts of these points are given in all treatises of medical electricity, but they may be found experimentally by using a fairly strong faradic current which gives a good contraction of a muscle on the normal side, say the biceps, and then moving the electrode over the whole muscle, when it will be found that at one or two points on the inner side of the biceps the greatest result is obtained—this then is the motor point. This place should be marked with an aniline pencil, and the electrode should be kept there, while the current is diminished by drawing out the secondary coil of the battery till a contraction is only just obtained; this gives the minimal stimulus to which the muscle will contract. The same should be done with the muscle of the opposite side. If there is any difference between the two sides, we must first see that this is not due to the different resistance of the skin. To ascertain this the constant current must be switched on, and using the same electrodes a current which will give a deflection of the galvanometer needle of, say, five milliamperes is kept on quite steadily over the motor point of the muscle under examination. The same current is then to be applied to the same point on the other limb, and the deflection of the needle noted; if this is less on this limb, it will show that the resistance is greater, and consequently a stronger faradic current would be required to produce a contraction on this side than on the other, though the muscle is quite healthy. If the resistance to the constant current on the two sides be equal, and yet the muscle on one side requires a stronger faradic current for its contraction than on the other, there is probably degeneration of the nerve or muscle, which requires the strong current for its contraction.

In testing with the constant current the same method is used as above. The positive pole, or anode, is to be connected with the large pad, and the negative, or kathode, with the smaller testing electrode ; the current should be made and broken by a contact breaker on the battery, which is better than having it on the electrode. Using at first a strong current to get a good contraction, this is gradually diminished until a contraction is only just obtained when the current is made, then, still leaving on the electrode, the amount of current is read off by the deflection of the galvanometer needle. After breaking the current, reverse the poles by the current reverser on the battery, and find out in the same way at the same spot on the muscle or nerve the deflection of the galvanometer required to produce a contraction with the positive pole, while making the current. It should be noted that, whereas with the faradic current a rapid series of contractions or tetanus of the muscle occurs, with the galvanic only a single contraction occurs at the moment the current is made, or when it is broken if the current be strong.

In an electrical examination the muscles and the nerves going to them should be tested in the manner already shown, and it will be well to state briefly the changes found in disease.

When a muscle is cut off from its trophic centre in the medulla or cord by a lesion of its nerve, as in a case of facial paralysis, the following *reaction of degeneration* is observed. *In the nerve* the faradic and galvanic reactions are gradually lost, and are not obtained at all in about two weeks' time. In the *muscles* (*a*) the faradic excitability is lost along with that of the nerve; while (*b*) to the galvanic current there is at first a diminished excitability, and then at the end of two weeks a marked increase up to the third or fourth week, and then it gradually diminishes. The quality also is altered, and in place of a brisk contraction rapidly passing off we have a slow, deliberate contraction, which sometimes persists all the time the current is passing. Serial changes are noticed, and in place of obtaining, as in health, the contraction at the negative make, or kathodal closure contraction (*i.e.* K.C.C.), as it is called, with a weaker current than at the positive or anodal closure of the current (*i.e.* A.C.C.), the reverse is obtained, the A.C.C. requiring an equal or even a less current to produce a contraction than the K.C.C.

In what is known as the *partial reaction of degeneration*, the reaction of the nerve is unaltered, while the muscle shows marked changes both in quantity and quality to the galvanic current.

While speaking about electricity, it will be well to mark a few general remarks about treatment.

Electricity is used for two chief purposes—(1) as a stimulating agent, (2) as a sedative agent. (1) In the former case the muscles, when they will react to faradisation, are treated by brushing them with one of the poles, the other being placed over the nerves going to these muscles; or if the muscles react only to the constant current, the positive pole is placed on the spine, and the negative is used for brushing the muscles, the strength being just enough to cause a well-marked contraction. This treatment of the muscles is called the *labile* method. In stimulation of the skin by faradisation, the dry wire brush should be used as one pole, the other being wetted and placed on the limb higher up. (2) For sedative action the constant current is used, and never the faradic; and whereas the negative pole is used for stimulation, the positive or anode is used for sedative action. The negative is placed on the spine with a large electrode, and the positive is placed on the affected nerve without any current, this is gradually turned on without any break and without moving the electrodes, so as to increase the current till a gentle burning is felt without producing any shock. This is called the *stabile* method.

Reflexes.—(1) *Deep* or *tendon.* Physiologists have differed as to whether these are true reflexes or not; but for clinical purposes it suffices to remember that they are lost when there is a lesion of the muscle or its motor nerve, of its trophic centre in the cord (anterior horn), or of the posterior cornu or root.

The *knee jerk* or patellar-tendon reflex is obtained by feeling first for the patellar tendon, and striking it with the edge of the hand or with a heavy stethoscope with india-rubber round the rim, when a contraction of the quadriceps ensues. The position of the limb does not matter, as long as the knee is bent and the muscles are *absolutely relaxed*; sitting on a table and letting the legs swing is a very good way. A feeble knee jerk may be increased by making the patient hook his fingers together and pull (Jendrassek's method); but it has no effect when the knee jerk is absent. When it is much increased, the slightest tap will produce a contraction, and then a *rectus clonus* in the form of a rhythmical contraction can be obtained when the knee is extended by suddenly drawing down the patella, and keeping some tension on it.

Ankle clonus is obtained by making a sudden flexion of the patient's foot, and then letting it rebound, but still keeping up some pressure, when a to-and-fro movement of the ankle joint ensues. It is best obtained when the knee is bent; and a different angle suits different cases. The knee jerk is present in health, but ankle clonus is not.

The *triceps jerk* is got by flexing the elbow, allowing the forearm to hang vertically downward, and striking just above the olecranon, when a contraction of the triceps occurs.

The *radius tap* is produced by striking smartly the styloid process of the radius when the muscles are quite relaxed and the elbow flexed. A contraction of the flexors of the elbow joint is thus produced.

The two last symptoms are often not obtained in health, and if well marked are evidence in favour of organic disease, especially lateral sclerosis. In trying ankle clonus great care should be taken that the muscles are relaxed, as there is a form of spurious, irregular ankle clonus in hysteria, where the patient voluntarily contracts the calf muscles.

The *lower-jaw jerk*, or even clonus, can be obtained, but not as a rule in health, by laying the forefinger flat just above the chin or on the teeth of the lower jaw, and tapping it from above.

(2) The *superficial reflexes* are true reflexes, obtained by irritating the skin with a quill pen. The most important from below upwards are the plantar, obtained by stimulating the sole of the foot; the cremasteric, causing elevation of the testicle when the inner part of the thigh of the same side is irritated; the abdominal and epigastric, causing the umbilicus and epigastrium to be drawn outwards when the skin in the lumbar and hypochondriac regions are respectively stimulated. In the head, the reflex contraction of the soft palate is obtained by tickling the fauces, and the conjunctival reflex of blinking by touching the cornea.

The organic reflexes.—The *vesical reflex* may be deranged so as to produce (1) incontinence, or (2) retention.

(1) Incontinence may be (*a*) *simple*, when the lesion destroys the vesical centre in the lumbar enlargement, and the sphincter being paralysed, the urine runs away as fast as it enters the bladder; or (*b*) *intermittent*, where the lesion, being in the dorsal region, cuts off the cerebral influence from the vesical centre, so that when the bladder is full the sphincter is reflexly relaxed and the urine discharged without the patient being able to prevent it, and if the sensory tract is also involved, the patient is unconscious of the act. (*c*) *Overflow incontinence* occurs where the muscular action of the bladder is weakened by the absence of voluntary power, the bladder becomes full, and relaxes the sphincter by pressure and the urine dribbles away.

(2) Complete retention is produced when the detrusor

centre for causing contraction of the vesical walls is paralysed, but not that for the sphincter; if not relieved, it may pass on to overflow incontinence.

Anal reflex.—Incontinence of fæces may be (1) simple, when the anal centre in the lumbar enlargement is destroyed and the sphincter relaxed; or (2) intermittent, when either the excreta are passed automatically without the patient being able to prevent them, or there is constipation, due to the lesion being in the spinal cord above the anal centre.

The condition of the sphincter ani can be ascertained by introducing the finger into the rectum.

SENSORY SYSTEM

In testing tenderness of the head by direct percussion, it is as well for the observer to tap his own head first, to ascertain how slight a tap causes dis comfort in health, and how carefully this method must be used.

Tactile sensation on one side of the body is best tested by touching similar places on the two sides very lightly with the observer's finger without moving it along the skin, or with a piece of wool, when the patient's eyes are closed, and testing the veracity of the patient by pretending to touch and not doing so. When both limbs are affected, the appreciation of a touch must be compared with a part, as the trunk or face, where sensation is normal. Often the slightest touch can be felt and localised, but the sensation is not a natural one as compared with another part; this may be called relative anæsthesia.

Painful impressions are tested by a pin, and more

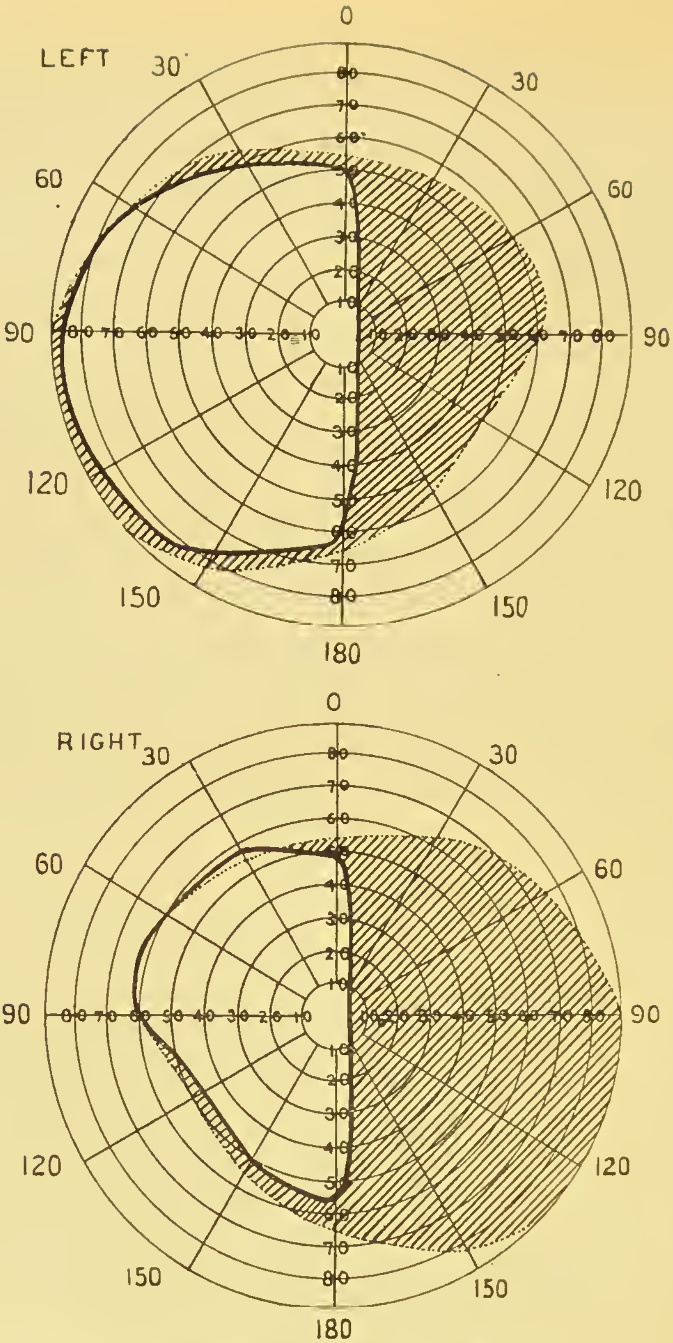

Fig. 18.—Perimeter fields of vision showing right hemianopia affecting both eyes, and due probably to thrombosis affecting the left optic radiations. The shading denotes the part of the normal field of vision where sight was lost.

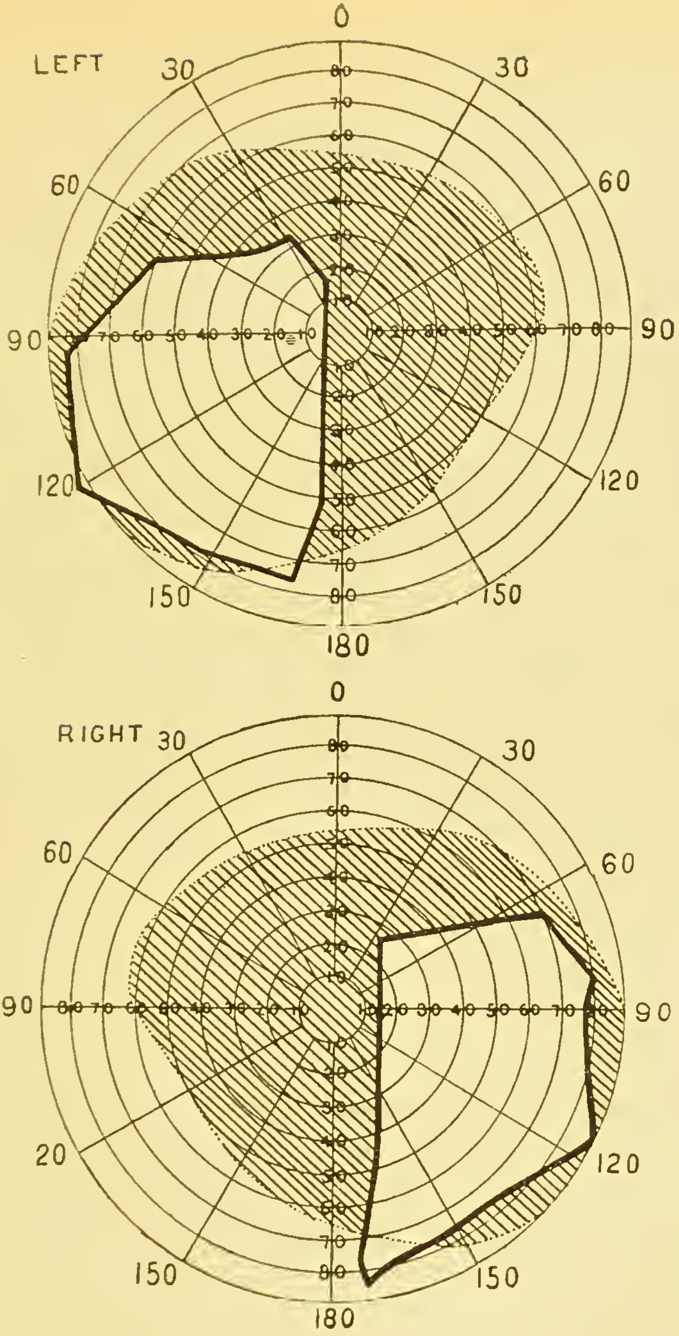

Fig. 19.—Perimeter fields of vision showing binasal hemianopia, in a case of locomotor ataxia, due probably to atrophy of the lateral fibres in each side of the chiasma, from a case of locomotor ataxia (Lang and Beevor).

accurately by Dr. Head's method of touching the part with the middle finger, and while this is in contact to slide a pin, held with the forefinger and thumb, along the middle finger, so as to prick the skin, thus obviating the fallacy produced by the difference in area covered by a pin point and the tip of the finger.

Sensation of temperature is best examined by two test tubes containing ice and hot water. Sensation is in some conditions delayed to painful impressions, and this is tested by pricking the part with a pin and noticing how many seconds before the patient says that he feels it. Loss of muscular sense is detected by putting the different joints in certain positions with the eyes closed, and asking the patient to describe them, or to put the other limb in a similar position; on opening the eyes patients can tell correctly if the joint is in a different position to what they thought. The appreciation of weights is tested by using balls of the same size, but differently weighted, and asking the patient to differentiate them.

Exact localisation of the place touched is tried by making the patient touch with his normal forefinger the exact spot on his arm or leg, which the observer has just touched when the patient's eyes were shut.

Organs of special sense.—*Sight* is to be tested for *acuity* and for colour vision in the manner described in ophthalmic works. The *field of vision* is also of the greatest importance, and can be tested roughly by holding up the two hands on either side of the patient's head, and ascertaining if he can see them both; or more correctly by making the patient close one eye and look at the observer's nose, who now makes his finger describe a quadrant of a circle from the side of the patient's ear forwards to the middle line, and noting the exact point where the finger is seen. The same mode of procedure is used for the opposite or nasal quadrant, and for the upper and lower vertical quadrants. In semi-comatose or aphasic cases hemianopia can be considered probable, if the patient does not blink when menaced by a blow on one side and does on the other. For taking an accurate chart of the field of vision, an instrument called a perimeter must be used. In examining such a chart we must note whether sight is abolished in both temporal halves of the visual fields (due to pressure on the middle of optic chiasma); in both nasal halves of the fields (due to a lesion on each side of the chiasma) (fig. 19); in the corresponding halves of each field of vision—*i.e.* temporal half of one eye, and nasal half of the other, called "homonymous hemianopia" (fig. 18) (due to disease of the opposite optic tract, and the rest of the visual path behind this as far as the occipital cortex);[1] or, lastly, if there is a concentric loss of vision which is greatest in the eye opposite to the lesion (probably due to a lesion in the angular gyrus or to hysteria). Besides the fields for white light, those for colours should also be taken when they are affected.

In noting *subjective sensations*, particular attention should be given to ascertain in which part of the field of vision they appear and, if they move, in which direction.

In ophthalmoscoping the optic discs (which should never be omitted), the presence of optic neuritis or atrophy should be observed. In *optic neuritis* the first change to be noted is a blurring or indistinctness of the edge of the disc, especially on the nasal side. This increases till all the edge of the disc is obscured, and the swelling spreads beyond the edge of the disc; the veins are enlarged and tortuous, and may be concealed by the swelling, especially at the edge of the disc. The swelling may end by subsidence and recovery, or pass on to atrophy. Optic atrophy occurs primarily in locomotor ataxia as grey atrophy, where the vessels are not much altered in size and the lamina cribrosa is visible; and secondarily to optic neuritis or pressure on the optic nerve, where the disc is finally quite white. After optic neuritis the vessels are very small, and there are often evidences of old retinitis, as white spots especially at the yellow spot, and the physiological cup is filled up by new tissue.

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[1] The reaction of the pupil to light thrown sideways on to the two halves of the retina should be compared, as in lesions of the optic tract, say the left, the pupils do not react to light coming from the blind fields of the right side.

Besides the optic discs the retina must be examined for albuminuric retinitis, syphilitic choroiditis, vascular changes as embolism of the central artery of the retina, and hæmorrhages in the retina occurring in albuminuria and severe anæmia. It must be remembered that optic neuritis may occur in anæmia.

Hearing is tested by a watch for aerial vibrations, taking care that the patient does not see the distance the watch is held; and for perosseal impressions by a tuning-fork placed on either mastoid process, and noting for how many seconds the observer can hear the fork on his own mastoid after the patient has ceased to hear it. Deafness from cerumen accumulations must be eliminated.

Subjective noises must be noted as to their situation in the head or in either ear; their character, hissing, roaring, etc.; whether persistent, intermittent, or pulsating, synchronous with cardiac beat or not, or increased by posture.

Taste for the different parts of the tongue—*i.e.* the tip, the edges, and the posterior dorsum—and the palate and its arches is tested by making the patient close his eyes and put out his tongue; a drop of glycerine, vinegar, a solution of quinine, or common salt, is dropped on the supposed affected side, and without withdrawing the tongue the patient is to hold up his hand as soon as he tastes. A good plan for testing taste, especially on the palate, is that by two or three cells of a voltaic battery, the insulated wires being twisted together so that the free ends are close together without touching, and these when applied to the tongue produce a sour taste when the current is turned on. Sweets and acids are tasted best at the tip and edges, and bitters at the back of the tongue, while there is an absence of taste on the rest of the dorsum.

CHAPTER V.

DISEASES OF NERVES.

UNDER the above heading will be described neuritis local and multiple.

LOCAL NEURITIS.

An inflammation of one or more nerves of a limb.

Causes.—Primary neuritis arises externally from exposure to cold, injuries, pressure, or contusions ; and internally from syphilis, gout, diabetes, or rheumatism. Secondary neuritis is due to pressure, as from tumours.

Any nerve may become affected, especially the sciatic nerve giving rise to sciatica, and the brachial plexus. Under the head of pressure we have the so-called "sleep palsies" from lying on the arm, and "crutch palsies" from pressure on the nerve trunks in the axilla. Injury to the roots of the brachial plexus by stretching, is caused especially by falls on to the head and shoulder, or occurs in children born with a breech presentation from traction on the shoulder.

With regard to age and sex, secondary neuritis occurs between forty and fifty, and sciatica is much more frequent in men than women.

Symptoms.—In the acute form constitutional symptoms, as slight fever, may be present, but the local symptoms are the most prominent, and consist at first of pain, which may come on gradually but more often suddenly, and shoots down along the nerve. The pain, which is at first only produced by movement, becomes later of a continual burning character, and is always increased by movement, and the nerve which can sometimes be felt to be swollen is very tender; this is especially so over certain points, as in the sciatic nerve by pressing over the sciatic notch and the popliteal space when the knee is flexed. This pain is followed by tingling and hyperæsthesia, less commonly by loss of sensation, and sometimes by paralysis of the muscles, in the part supplied by the nerve, especially in the case of "sleep" and "crutch" palsies. The acute stage lasts for two or three days, and then passes on to the chronic, when the constitutional symptoms disappear. In the chronic stage there is still pain and tenderness in the nerve, and in some cases loss of sensation of the skin area supplied by it, and paralysis and wasting of the muscles, which in a week or two may show the reaction of degeneration (p. 73). Trophic changes are apt to occur, such as a glossy appearance of the skin, and joint changes.

Sciatica often begins with pain in the back, as lumbago, which is much intensified by any movement putting the lumbar muscles into action as stooping down with the knees straight, and the pain subsequently passes down the sciatic nerve of one side, but very rarely are both legs affected. In sciatica the patient keeps the knee flexed and the hip extended to prevent tension in the nerve. In "sleep palsies," produced by lying on the arm during sleep, the ulnar nerve is the most commonly affected, and here pain is slight but numbness and anæsthesia are well marked, and also paralysis of the muscles supplied by the nerve.

Secondary neuritis is due to pressure of tumours, aneurysms, dislocation about the shoulder, callus of fractured bones, abscess about the hip joint, and sacro-iliac disease. And these possible causes should always be looked for. There is less tenderness in the nerve trunks, and the symptoms are more gradual than in the primary form.

Pathology.—An inflammation of the nerve sheath, called perineuritis, occurs in slight cases, where the sheath is swollen and red; or the myelin white substance is inflamed and breaks up into globules, and the axis cylinder may disappear in severe cases where there is marked anæsthesia and paralysis and wasting of the muscles.

Diagnosis has to be made from simple neuralgia, and in the case of sciatica from hip-joint disease.

The nature of the neuritis has to be ascertained, whether primary, from injury or cold, or from syphilis, gout, or diabetes; or secondary to pressure.

The diagnosis of a neuritis depends on pain and tenderness along a definite nerve trunk with anæsthesia and loss of power in the parts known to be supplied by such a nerve. In neuralgia the pain is more paroxysmal and intermittent, it is increased less by movement, and it is not associated with anæsthesia or paralysis of the muscles. Secondary neuritis of the sciatic nerve is due to pressure in the pelvis, and the characteristic tenderness of the primary form is often absent in the sciatic nerve when the pain persists. It has to be diagnosed from tumours of the cauda equina, where the pain is usually bilateral and the sphincters are affected.

Diseases such as gout, syphilis, and diabetes must first be eliminated before the neuritis can be put down to catching cold only. Injury of the roots of the brachial plexus is distinguished from that of a peripheral nerve, like the median, by the group of muscles affected —the deltoid biceps and supinator longus being especially involved together—and the area of anæsthesia (see table and diagram at end of book), being those corresponding to the distribution of particular roots and not to that of a peripheral nerve. The same holds good with regard to lesions of the lumbar or sacral plexuses.

Prognosis.—A slight attack from cold may be acute for from two to four weeks, and may then subside, or pass into a chronic condition, which may last several weeks or months. As a rule, the severer the initial attack the longer will the disease last. *Traumatic* neuritis and "sleep palsies" are the most favourable. The prognosis is necessarily bad when neuritis is caused by tumour or diseased bone which cannot be removed.

Treatment.—Any cause of pressure should be removed, such as a growth pressing on a nerve; and in cases where a history of gout, syphilis, or diabetes can be made out, the appropriate treatment for these should be given. The general treatment of a case brought on by cold consists in giving a purgative at first and then diaphoretics, as liq. am. acetatis and citrate of potash in full doses; absolute rest is imperative, and the limb should be placed in the position most comfortable. Locally the pain is best relieved by a liniment composed of equal parts of linimentum belladonnæ, lin. chloroformi, and lin. aconiti, rubbed in gently until tingling is produced, and by linseed poultices or hot fomentations applied over the painful part. If the pain is not relieved, hypodermic injections of morphia, beginning with $\frac{1}{6}$ grain, or hydrochlorate of cocaine, gr. $\frac{1}{10}$ to $\frac{1}{2}$, may be given, but these remedies should only be used for excessive pain, and not habitually. During the acute attack a grain of blue pill once or twice a day has the most influence on the inflammation (Gowers).

In the chronic state counter-irritants, as mustard plasters placed along the whole length of the nerve, or blisters, should be used; or electricity in the form of the constant current, but not the faradic, should be applied with the negative pole to the spine, and the positive over the lower end of the affected nerve, and the current should be gradually increased without breaking the circuit till a gentle burning is felt in the nerve.

If there is marked wasting of the muscles, they should be treated by brushing them with the negative pole of the constant current of a strength just sufficient to make the muscles move, and by massage.

In chronic cases of some months' standing, and especially in cases of sciatica, in which a dull aching pain comes on after exercise, a course of Turkish baths gives the best results, and the main point is, that after the shampooing the strongest douche should be played over the nerve (the back of the thigh in sciatica) with warm water gradually cooled down to cold.

MULTIPLE NEURITIS.

This is called also peripheral neuritis, polyneuritis. A symmetrical inflammation of several of the peripheral nerves, either simultaneously or in rapid succession.

Causes may be grouped as: (1) poisons from without—(*a*) alcohol, (*b*) lead; (2) poisons from within, as leprosy and beri-beri, and after septicæmia, diphtheria, small-pox, measles, etc.; (3) rheumatic from cold. Of these the most important is the first, the alcoholic. Beri-beri is an endemic disease occurring in the East, which is rarely seen in England, and is then imported.

Alcoholic Neuritis.

This is the form most often seen.

Etiology.—It occurs more frequently among women, and among those who habitually drink spirits continually rather than among men who occasionally get drunk. The most common age is between thirty and forty, but it is met with at any age between twenty and sixty.

Symptoms.—The onset is as a rule not sudden, but begins with a feeling of numbness and pins and needles in the hands and feet, followed by sharp pains, and accompanied by extreme tenderness in the nerve trunks, muscles, and skin; there is often in a day or two some inco-ordination in the limbs, and then a failure of muscular power. The typical places for this loss of power are the muscles on the outer side of the leg and on the back of the forearm, supplied by the external popliteal and posterior interosseus nerves respectively. Consequently the patient has dropped-wrists and dropped-feet, and finds he is unable to extend the toes, to dorso-flex the ankles, to invert or to evert them; also in the upper limb, he cannot extend the fingers, the thumb, or the wrist. Almost invariably the corresponding parts of the right and left sides are affected at the same time, and the symmetrical flexion of the fingers and wrists and the extension of the ankles with the foot in a line with the tibia are very characteristic (fig. 20).

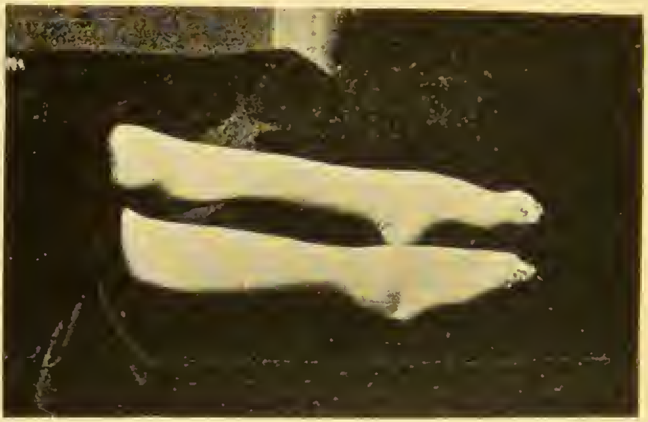

Fig. 20.—Typical position of the feet from a case of multiple neuritis affecting all the limbs, showing the dorsum of the foot in a line with the tibia. From a photograph.

On the sensory side, besides the tingling and extreme tenderness which are very characteristic, there is some anæsthesia of the hands and feet, which spreads up the outer side of the leg and the radial border of the forearm.

Inco-ordination of the arms and legs is sometimes present with more or less sensory symptoms and without any marked loss of power.

In the further progress of the disease, the other muscles below the knee and elbow are affected, the extensors and then the flexors of the knee and elbow may be paralysed, and subsequently the muscles of the hip and shoulder. In very severe cases, the trunk muscles, the intercostals, the diaphragm, the heart, and very rarely the face and tongue, are involved. The muscles waste very much, and in two or three weeks' time electrical changes occur, so that they cease to contract to the faradic current, or only to the strongest one, and give the reaction of degeneration with the constant current, while the nerves lose all response to electricity.

If only one set of muscles, *i.e.* the extensors, be paralysed, the unopposed flexors will produce a contracture with deformity of the joint.

The knee jerks are lost if the disease involves the extensors of the knees, and also even when there is only slight impairment of power with tenderness to pressure in the muscles; the loss of the knee jerk is therefore a very frequent symptom.

The plantar skin reflex is as a rule lost, but in early or slight cases where there is hyperæsthesia it may at first be well obtained.

Sensory disturbances increase along with the motor, which they often exceed in extent, and the limbs and their extremities are affected in a particular manner, so that a slight touch is not felt, but firm pressure over the muscles and the nerve trunks gives rise to acute pain. Analgesia or loss of sensation to painful impressions is rarely present, but a delay of a few seconds may occur in appreciating them. Besides the tenderness, there are acute boring, burning pains in the nerve trunks.

The tenderness of the muscles is most characteristic, and is well shown by pressing the skin over the anterior tibial muscles and comparing the excessive pain thus produced with the slight discomfort of pressing the skin against the anterior surface of the tibia.

Trophic changes occur sometimes in cases of long standing, in the skin, nails, and joints, similar to those occurring in local neuritis; and œdema may be found in the lowest parts of the paralysed limbs.

The sphincters are very rarely involved, and consequently the patient in nearly all cases can hold and pass his water and motions; the rare cases in which they are affected is probably due to spreading of the disease to the cauda equina or to the spinal cord.

The *course* and *duration* vary very much; but in an average case the disease increases from two to four weeks, then remains stationary for a month or two, and then begins to improve. Spontaneous pain becomes less, but the tenderness still persists; muscular improvement occurs in the inverse order of onset, or in other words the muscles which are the first and the most affected are the last to recover. In very acute cases the respiratory muscles may quickly be involved, and cause death either directly or by pneumonia. Death may also be caused by other diseases due to alcohol, as cirrhosis of the liver, Bright's disease, chronic cerebral meningitis, etc., or from phthisis, a frequent complication. Mental affections, as delirium tremens, may usher in the neuritis.

Pathological changes are found in the affected nerves, and particularly in the nerve fibres themselves as opposed to the connective tissue. In recent and severe cases the nerve fibres are reddened and swollen, and on microscopical examination acute degeneration of the individual fibres is very conspicuous. The myelin of the nerve is disintegrated and broken up, the axis cylinder has disappeared, and the connective tissue between the bundles of fibres is slightly increased. The changes in the nerve fibres are especially well brought out by staining with osmic acid and mounting in glycerine, when the collections of the broken-up myelin form numerous black points. As the symptoms showed, the changes are more marked in the peripheral parts of the nerves than towards the spinal cord, and the nerves most severely affected are the radial and the anterior tibial. The muscles supplied by the affected nerves are atrophied, they become pale, and in severe cases the fibres lose their striated appearance and show signs of fatty degeneration. Generally the spinal cord is not affected, but in some cases signs of chronic meningitis and diffuse myelitis have been found. The main change is therefore in the nerves themselves, and this would explain the severe pain, tenderness, and anæsthesia, the loss of power and wasting of the muscles with the electrical changes. The fact that the nerve fibres are affected more than the sheath explains why the pain and tenderness are so much more marked here than in ordinary local neuritis. The symmetry of the disease is referred to the toxic effect of alcohol on the blood, and the peripheral character to the theory that the vitality of the nerves and their capability of resisting disease are lessened the farther the distance from the trophic centres in the spinal cord (Gowers). The extensors of the fingers and toes suffer more than the flexors, probably because they are the weaker muscles whose function is to put the joints in the best position for the flexors to work.

Diagnosis.—See p. 187.

Prognosis.—This differs very much in the different stages of the disease. At the onset, while the symptoms are still progressing, it is not possible to say at what point they will stop, and therefore it is not possible to give a definite prognosis. As death from suffocation is most to be feared, the involvement of the muscles of the trunk, and especially the intercostals and diaphragm, is very serious. A fatal termination is most likely to occur in the early days of the disease from a rapid extension of the disease to the respiratory muscles or to the heart through the vagus nerve. When the disease ceases to progress the prognosis is much more favourable, and death does not occur except from other diseases. Any alteration can hardly be expected for a month or so, when probably improvement will begin. This is exceedingly slow, and takes months before the muscles recover their power. Six months or a year after the onset, improvement in the paralysed muscles may still be expected as long as other muscles are recovering. In some cases recovery does not take place, and in these the spinal cord has been considered to be affected (Gowers). As a rule sensation returns before motion; pain and anæsthesia pass off, but tenderness to pressure often persists even during the stage of improvement. The return of the faradic electric excitability is a very good sign, but occasionally power returns before the faradic excitability. Generally speaking the prognosis is worse the more acute the onset and the greater the extent attacked.

Treatment.—The first thing is to prevent the cause, and a thorough investigation should be made as to alcoholic excess. This is often very difficult, especially with regard to women, and in any doubtful case the patient must be removed from her friends and put under the care of a trustworthy nurse. Unless the pulse becomes very weak, alcohol should be stopped altogether.

In the acute stage the patient should be kept in bed with absolute rest, the affected limbs being put in a position most comfortable to the patient, and at the same time in one in which deformities will not be produced by contraction of unopposed muscles. A sandbag against the sole of the foot will keep the extended ankle at a right angle and prevent it becoming a permanent deformity. Pain should be met with anodyne liniments, as equal parts of lin. belladonnæ, chlorof., and aconite, and hot fomentations and linseed-meal poultices over the painful nerves. Hypodermic injections of morphia $\frac{1}{6}$ gr., or cocaine $\frac{1}{10}$ to $\frac{1}{2}$ grain, may be required, but should be sparingly used. Extr. cannabis Indicæ (Indian hemp), gr. $\frac{1}{4}$ to 1 in pill, antipyrin gr. v to xv, exalgine gr. ij to vj are also useful in allaying severe pain; while to procure sleep where there is restlessness without much pain, sulphanol in 10 to 30 grains, or bromide of ammonium in ʒj doses.

The general treatment in an acute case consists in promoting general diaphoresis and diuresis by liq. am. acetatis, pot. citrat., spir. æth. nitrosi; if the attack be due to cold, sodic salicylate 10 to 20 grains every two or three hours is useful at the onset.

During the stationary and improvement stages, tonics such as quinine, iron, strychnine, and cod-liver oil are required.

Electricity, which is so useful as a means of diagnosis, should not be used till the muscles show signs of recovering; the constant current should be employed, the positive pole being placed over the lumbar region of the spine, and the negative with a large electrode gently brushed over the affected muscles, the current being just strong enough to produce a contraction of the muscles without causing pain. Massage and rubbing of the muscles and manipulation of the joints should be used at the same time, but not till the muscles show signs of returning power; for it is not conceivable that either electricity or massage can accelerate recovery in the nerves—all that they can do is to keep up the nutrition of the muscles supplied by these nerves.. This treatment may be persevered with even after six or twelve months with a hope of further improvement as long as the muscles react to the constant current.

Neuritis from poisoning by lead is described further on. Of neuritis from poisons from within, beri-beri and leprous neuritis are examples.

Beri-beri and Leprosy.

Beri-beri is an endemic neuritis occurring in the East Indies and Japan, and occasionally cases are brought to this country. It is a disease which is propagated through communities who have been repeatedly exposed to the infection.

Symptoms.—The disease exists in two forms, the dry and the dropsical. The first symptoms are numbness and weight in the legs, some anæsthesia, weakness in the peroneal muscles and dorso-flexors of the ankle, which very early show signs of electrical degeneration. Along with the loss of power there is anæsthesia of the parts supplied by the affected nerves with tenderness of nerves and muscles. The chief difference between this disease and other forms of neuritis is the readiness with which the cardiac branches of the vagus nerve are affected; the action of the heart becomes rapid and irregular, and dropsy occurs, which may affect the legs and the serous cavities of the pericardium, pleura, and peritoneum, and this implication causes the dropsical form of the disease.

In severe cases the other muscles of the legs may be affected, the arms, trunk, diaphragm, and even the face and larynx.

Death may occur from heart disease, and sometimes very rapidly after the disease has lasted a week or so; but the symptoms may last several months after the acute stage has passed off.

Pathology.—The changes are entirely in the peripheral nerves, and are considered to be due to a specific organism; the medullated white substance of the nerves becomes degenerated, and finally broken up with disappearance of the axis cylinders.

Treatment consists in removing the patient from the infected district, which is often sufficient for improvement, and treating the cardiac trouble with digitalis and strychnine.

The other form of endemic neuritis due to *anæsthetic leprosy* may be briefly mentioned. Leprosy occurs in the

East, and in certain parts of Europe, notably Norway. It takes years to develop, and appears to be inoculable by long residence with affected people, and to be due to a specific organism. The symptoms consist in slow muscular wasting with electrical changes, especially of the hands and feet, and with some loss to all forms of sensation in irregular patches. Trophic lesions are very common, and the extremities ulcerate and "drop off." The lesion in the nerves is a fibrous overgrowth of the sheaths of the nerve bundles, and the nerve fibres themselves are secondarily compressed. This would account for the irregular distribution of anæsthesia, which does not correspond with that of the whole nerve trunk. In this fibrous tissue in a recent case has been found the leprosy bacillus.

The atrophy of the small muscles of the hands, the irregular patchy areas of anæsthesia, and especially the ulceration and mutilations, are unlike any other disease, the last symptoms being more extensive than are seen in syringo-myelia (*q.v.*), which leprosy most resembles.

CHAPTER VI.

DISEASES OF THE MEMBRANES OF THE SPINAL CORD.

THE cerebro-spinal axis is covered by three membranes, of which the outer or *dura mater* is a strong, inelastic, fibrous sheath. Its inner surface is smooth and lined with epithelium, while its outer is rough and adheres closely to the cranium forming its periosteum, and is loosely connected to the vertebral canal with fat and connective tissue. At each intervertebral and cranial foramen it is continued as a prolongation on the nerve, and is merged into the nerve sheath. It extends down to the second sacral vertebra. It forms the falx cerebri, the falx cerebelli, and the tentorium cerebelli, and is composed of two layers, between which are the different venous sinuses.

The *pia mater* is a delicate, fibrous, vascular membrane on the brain; but on the cord it is thicker, less vascular, and more adherent to the subjacent cord, and its increased thickness is due to an external layer of longitudinal bundles. At the roots of the nerves the pia becomes continuous with their connective-tissue sheaths. The pia mater is prolonged as a fold down the anterior and posterior fissures of the cord, and dips into all the fissures and sulci on the surface of the brain. It also forms the velum interpositum, which is prolonged into the lateral ventricles through the transverse fissure of the brain.

The *arachnoid* is a delicate membrane which does not dip down into the fissures. It consists of ribandlike bundles of fine fibrous tissue interlaced in several layers, and lies between the dura and pia. Between it and the pia is the subarachnoid space containing cerebro-spinal fluid; at the posterior part of the spinal cord a membranous partition passes in the median plane from the pia mater covering the posterior median fissure of the cord to the opposite part of the loose portion of the arachnoid—it is called septum posticum, and contains blood vessels. The subarachnoid space is well marked at the base of the brain and in the spinal cord, and extends over the interpeduncular space as far forwards as the optic nerves, and behind it forms an interval between the cerebellum and the back of the medulla. It communicates with the ventricles of the brain by the foramen of Majendie—the opening into the lower part of the fourth ventricle through the pia-matral expansion which covers it—and it is continued below into the subarachnoid space of the cord. The nerves issuing from the cord and brain receive their perineural covering from the pia mater, as well as two looser sheaths, an outer from the dura mater and an inner from the arachnoid. A certain quantity of fluid exists between the dura and arachnoid, but not much.

The diseases of the spinal membranes which will be described here are inflammation or meningitis, and hæmorrhage or hæmatorhachis; the diseases of the cerebral membranes are described further on.

INFLAMMATION OF THE MEMBRANES OF THE CORD, OR MENINGITIS.

As there is some difficulty with regard to the terms used, it will be best to follow the plan used by Sir W. Gowers, and subdivide meningitis into two forms: (1) that which begins outside the dura mater, external meningitis; (2) that which begins within the dura mater, internal meningitis.

External Meningitis.

This is also called external pachymeningitis.

Causes.—It is usually secondary to disease of the bones of the spine, such as caries, and in rare cases to syphilis. It appears sometimes to occur as a primary acute disease in weak subjects, but it is not common.

Symptoms.—In the primary disease there is pyrexia, rigors, and sweating; the spinal nerves become involved, causing pain, which radiates along the nerves and is increased by movement. Irritation of the nerves also causes spasms of the muscles and hyperæsthesia. As the disease advances, and if the inflammation becomes purulent, there may be motor and sensory paralysis with loss of reflexes and paralysis of sphincters. The symptoms are very like those of acute internal meningitis.

The secondary chronic form is rather an exacerbation of the disease of the spinal bones, the symptoms being especially pain in the back and along the nerve trunks, with signs of compression of the cord.

Pathology.—In slight cases the external surface of the dura mater is reddened and covered with lymph; in more severe cases, especially associated with caries, the membrane is covered with a layer of caseous matter, and in some cases this is purulent. In the primary form the whole space between the bones and membranes may be filled with pus, while in that secondary to caries, only the part of the membrane contiguous to the diseased bone is affected.

Prognosis in the acute form is very unfavourable as regards recovery and life, while in the secondary form the prognosis depends more on the amount of compression produced by the disease of the spine.

Treatment for the secondary chronic form is that of the spinal caries, with this difference—that whereas caries can in many cases be treated by rest, here, when the presence of external meningitis with deposits of caseous matter can be made probable, the operation of laminectomy with removal of these products of inflammation should certainly be recommended, and in all cases where the presence of pus is suspected it should be let out.

The treatment of the acute external is the same as that for internal meningitis (p. 105).

INTERNAL MENINGITIS.

This, which is called also leptomeningitis, begins within the dura mater, and occurs as an *acute* form affecting the pia mater and arachnoid, or as a *chronic* form involving the inner surface of the dura mater in addition to the pia and arachnoid.

Acute Internal Meningitis.

Causes.—Predisposing conditions are a tubercular history or bad feeding; and exciting causes are cold and injury to the spinal column. The disease is sometimes secondary to cerebral meningitis. A severe form occurs after septicæmia due to organisms, or after the acute specific fevers. A third form is that known as epidemic cerebro-spinal meningitis, which will be described later.

Symptoms.—These are due at first to irritation, and later to paralysis, of the motor and sensory roots of both sides passing through the membranes, and are sometimes preceded by general malaise and depression, headache and restlessness.

They begin with a rigor followed by pyrexia and intense deep-seated *pain* in the spine, being most acute over the particular part of the membranes affected, and increased by movement of the spine or external pressure. The pain has exacerbations of violent shooting along the nerve trunks in connection with the affected roots.

The muscles of the back are in a state of tonic spasm and rigidity, which begins in the muscles supplied by the affected part of the cord; the condition is due to direct irritation of the motor nerves, or to reflex irritation of the sensory nerves passing through the inflamed membranes. The spasm is often marked in the neck, producing retraction of the head or arching of the back, and it may extend to the limbs. On the sensory side there is extreme tenderness and hyperæsthesia in the skin and muscles supplied by the roots coming from the affected region of the cord. Reflex action is at first increased, and the sphincters of the bladder and rectum are affected with spasm, causing retention and constipation. Implication of the cerebral membranes is shown by headache, delirium, and coma.

The symptoms of paralysis which come on after those of irritation are loss of power and flaccidity in the muscles and anæsthesia in the skin in the same parts where previously existed spasm and hyperæsthesia. The pupils may be contracted, dilated, or uneven, if the disease is in the upper dorsal region. The arms or legs are most affected according as the disease is more marked in the brachial or lumbar enlargements of the cord. Paralysis of the vaso-motor nerves is shown by the *tache spinale*, the slow development and the persistence of the red streak produced by scratching the abdomen with the finger-nail. Reflex action is lost with incontinence of urine.

The case may end in deep coma with a high temperature; or death may ensue in a few days from asthenia or paralysis of the respiratory muscles with Cheyne-Stokes breathing; or the patient may get bed sores, and die in two or three weeks. Slight cases may improve, but with wasting of muscles and paralysis of motion and sensation, from which the patient takes long to recover.

Pathology.—Three stages are described. (1) The pia mater is congested with hæmorrhagic extravasations and swelling of the tissues, the spinal-cord surface and the arachnoid are also congested, while the cerebro-spinal fluid begins to be turbid. (2) The pia mater is much swollen, gelatinous with a purulent exudation and turbid cerebro-spinal fluid, and the exudation forms a layer over the cord and along the nerves. The cord may show signs of myelitis. (3) If absorption does not take place, the spinal membranes become thickened with adhesions, the cord becomes sclerosed or atrophied, and excess of fluid remains in the subarachnoid space.

The above description applies to simple meningitis. In the tubercular form these symptoms are not quite so severe, and pathologically the exudation is less and grey granulations are found in the membranes.

Diagnosis.—This has to be made from other forms of spinal disease (see p. 186). Arching of the back occurs in tetanus and in strychnia poisoning, but in these there is no pyrexia at the onset. Of the different forms of meningitis, the tubercular spinal form is usually secondary to cerebral meningitis.

Prognosis is always serious, and is worse in tubercular than in traumatic cases, and in young rather than in middle-aged cases. High temperature, early paralysis, signs of exhaustion are very unfavourable symptoms.

Treatment.—The patient should be kept absolutely at rest in a quiet, darkened room, and should lie in the position which is the most comfortable, and if possible on his side. Local treatment should be employed in the form of blood-letting, leeches, or dry cupping along the spine, followed by hot fomentations.

Internally the bowels should be freely opened by pulv. jalapæ co. gr. xx or calomel gr. v. The remedy which has appeared to give the best results is mercury, which is best administered as a daily inunction along the spine of ʒss. of the unguentum or oleate of mercury, and it should be continued until the gums just begin to be tender. Morphia given hypodermically, or chloral and bromides, is required for alleviating pain. Warm baths or hot-air and vapour baths sometimes give great relief and induce sleep, especially in simple cases due to cold.

In the chronic condition counter-irritation to the spine by sinapisms and blisters, and internally tonics as iron, quinine, and strychnia, are of service ; and for the muscular wasting and contractions left by the disease electricity and rubbing are required.

Chronic Internal Meningitis.

This exists in two forms, according as it begins in the inner layer of the dura mater, or the pia mater and arachnoid. The inflammation may be local or general.

Causes.—The disease occurs most often in adult age, and affects men more than women. Of important exciting causes are frequent exposure to cold, alcoholic excesses, syphilis, tubercle, and local injuries as falls on to the back or compression of the cord from diseased bone.

Symptoms.—Pain in the back comes on gradually with stiffness in the neck, and is increased by movement and pressure. Associated with this symptom are sharp, burning pains shooting along the nerve trunks arising from the affected part of the spine, and subjective sensations of numbness and tingling with hyperæsthesia in the parts supplied by these nerves. These symptoms persist for some weeks, and are followed by atrophy and loss of power in the groups of muscles supplied by the different roots of the spinal cord which are involved, together with loss of sensation in the skin supplied by these roots. The wasted muscles give the electrical reactions of degeneration, and if the lumbar region be involved the deep reflexes are lost and the sphincters paralysed.

When the disease begins in the pia and arachnoid, the signs of meningeal irritation—pain and rigidity in the back with excessive sensibility in the skin—are most prominent; whereas when the inner surface of the dura is first affected, the roots suffer most, and in consequence the radiating pains with subsequent paralysis are most characteristic.

A form of the latter disease has been described by Charcot and Joffroy as occurring especially in the cervical region, under the name of cervical hypertrophic pachymeningitis, in which there are stiffness and gradual pains in the cervical spine shooting down both arms, and followed by atrophy and paralysis of muscles supplied by the lowest roots of the brachial enlargement—*i.e.* the small muscles of the hand and the flexors of the wrist and fingers—giving rise to a peculiar deformity of both hands, the fingers being flexed, but the metacarpo-phalangeal joints and the wrists being hyper-extended; associated with these symptoms there is general weakness of the legs from compression of the cord.

Of the subsequent course of the different forms of chronic meningitis the prognosis is unfavourable, the most hopeful being in those due to syphilis or to traumatic causes.

Pathology.—The appearances differ according as the disease begins in the pia mater or in the inner surface of the dura. In the former the pia and arachnoid are thickened and inflamed, and on microscopical examination enlarged vessels and lymphoid cells are seen. The spinal cord is softened and by extension is infiltrated with lymphoid cells in the neighbourhood of the inflamed membranes, which gives rise to secondary degenerations, usually ascending. The inner surface of the dura is opaque, and the spinal fluid is increased and turbid. The pia may be extensively affected. On the other hand, when the disease begins on the inner surface of the dura—the hypertrophic internal meningitis of Charcot and Joffroy—the disease is more limited in extent. The dura mater, especially in the cervical region, is very much thickened and is composed of several layers, and sometimes forms a tumour-like appearance, which surrounds and compresses the nerve roots and the cord, producing atrophy and degeneration of the former, and myelitis in the cord.

Another form of local meningitis affecting the pia and arachnoid is due to syphilis.

Treatment consists in absolute rest, with counter-irritation to the spine by blisters, sinapisms, or the actual cautery. Mercury should be given internally, or the oleate may be rubbed into the back along the spine, and subsequently iodide of potassium given internally. Electricity, especially the constant current, and massage are needed for the wasted muscles, and later, tonics and cod-liver oil.

HÆMORRHAGE INTO THE SPINAL MEMBRANES, OR HÆMATORHACHIS.

Effusion of blood into the membranes of the spinal cord may be extra-meningeal between the dura mater and the bones, or intra-meningeal; and this latter is divided into subdural and subarachnoid according to the situation.

Causes.—Injuries such as fracture of the spine and blows on the back, and occasionally severe convulsions, have produced rupture of a vessel, causing especially extra-meningeal hæmorrhage. It may also occur in new-born children from injury at birth.

Symptoms.—The onset is very sudden, with severe pain in the back radiating along the nerve trunks of the limbs, associated with violent muscular spasms in the back and limbs, and tingling with hyperæsthesia, due to the irritation of the nerve roots and membranes. The urine is usually spasmodically retained. After the spasm follows paralysis of motion, and to a less extent loss of sensation, in the parts which were the seat of muscular contraction.

Unless the shock is very great, consciousness is not lost. The particular muscles affected with spasm will vary according to the part of the cord involved. The temperature is not as a rule raised, unless secondary meningitis occurs. Death very often occurs in a few hours; and if the patient survives, the paralysis persists some time.

Pathology.—The origin of the blood in extra-meningeal hæmorrhage is from the plexus of veins surrounding the dura, and in the intra-meningeal form from the vessels of the pia. The cerebro-spinal fluid may be blood-stained, and the cord may be damaged in the latter variety.

Treatment.—Absolute rest with the patient lying on his face is essential, with local abstraction of blood by leeches, wet cupping or scarification over the seat of pain, followed by the application of ice and giving ♏iij to ♏viij of the injectio ergotini hypodermica. If the diagnosis can be made with any certainty, there is no reason why the spinal canal should not be opened and treated surgically like hæmorrhage from any other part, provided that the patient can bear the shock of the operation; and in any case the washing out of the effused blood from the canal would give the nerves a better chance of recovery.

CHAPTER VII.

DISEASES OF THE SPINAL COLUMN.

CARIES OF THE SPINE (CALLED POTT'S DISEASE).

Caries of the spine only comes within the domain of nervous diseases so far as it affects the spinal cord and its roots by compression.

Causes.—In most cases there is a distinct tubercular history, and the condition seems to be started in many cases by injuries or strains of the spine.

Age.—It occurs most commonly in childhood or youth, but no age is exempt.

Symptoms.—Pain, especially on movement, and tenderness to pressure at definite vertebral spines are the first symptoms.

The deformity of the spine comes on gradually, and usually consists of a definite projection of two or three vertebral spines, one being often more prominent than the others, so that the finger passed along the spines "comes to a dead stop" at this prominent spine. In the cervical region the deformity is not so common, but in its place there is great thickening of the tissues.

The membranes and nerve roots of the cord are the first to be affected, and give rise to pain radiating along the intercostal nerves or along the nerves of the limbs with hyperæsthesia in the skin supplied by these nerves ;

but these are not so marked as the symptoms due to compression of the cord itself.

The curvature, which is most frequently angular, usually precedes by some months the cord symptoms, which gradually come on with motor paralysis.

Both legs are usually affected simultaneously, but sometimes one is before the other. The character of the form of the paralysis differs according to the position of the lesion, and whether the cord is compressed at the lumbar or brachial enlargement, at the mid-dorsal or upper cervical regions. At the seat of lesion the cells of the anterior horns will probably be affected and produce wasting and atrophy of the muscles they supply, and also the conduction of motor impulses along the lateral columns will be prevented, and the parts below the lesion will be paralysed. Hence if there is any pressure on the brachial or lumbar enlargement, there will be paralysis of the muscles of the arm or leg respectively with extreme wasting and electrical changes.

Most commonly the curvature is in the mid-dorsal region, and there is then gradual paralysis of the legs without wasting, but with increase of the tendon reflexes and rigidity of the legs. Sensation may also be affected for touch or pain and temperature, or rarely completely lost for all forms. The upper limit of the anæsthesia extends like a belt round the trunk, and the level corresponds to the area of the skin supplied by the particular segment of the cord compressed. Loss of sensation is less common, and occurs later than loss of power. The sphincters may or may not be paralysed. The superficial reflexes are lost at the level of the lesion and are increased below it.

In rare cases the symptoms come on in a few hours from sudden displacement of the bones. As complications may be mentioned bed sores and cystitis.

The **course** of the disease is very variable, and depends not so much on whether the bone disease is arrested as on whether the pressure on the cord is diminished. In some severe cases the myelitis spreads to other parts of the cord either ascending or descending. Improvement often occurs with rest, with return of sensation and some power, but with rigidity of the legs.

Pathology.—The condition is caused by tuberculous disease of the bodies of the vertebræ and the intervertebral substance, which are destroyed and fall together, giving rise to the angular projection of the vertebral spines. The cord is compressed by the inflammatory products of the disease, which may surround the cord outside the thickened dura mater and become purulent or caseous; less frequently the cord may be mechanically nipped over a projecting piece of bone. From this there ensues a slow, gradual compression, or a more or less rapid inflammation of the cord. The nerve roots are liable to be compressed or irritated by the bones or thickened membranes, giving rise to the sharp pains occasionally met with.

Diagnosis.—See p. 188.

Prognosis is more favourable than many other forms of spinal disease, and recovery may occur months after a complete loss of motion and sensation. This is more often seen in children. As it is not possible to know the extent of the tubercular disease, one cannot say with any certainty which particular case will recover; and it is difficult to tell how much of the symptoms is due to myelitis and structural changes in the cord. Prognosis is worse if the respiratory muscles are affected, or if there is tubercular disease elsewhere.

Treatment.—The first most important thing is *complete rest.* This is best effected by keeping the patient lying down flat on his back, and if the disease is in the cervical region fixing the head and neck by means of sand-bags. Combined with this, *extension* may be used, the upper part of the body being fixed by straps round the axilla or round the chin and occiput to the head of the bed, and extension weights of from five to ten pounds or more being applied over pulleys to the feet. Care should be taken that the spine is never rotated, and the patient should, when required, be rolled on his side by lifting up the two ends of one side of the sheet, to ensure the shoulders and pelvis moving at the same time. Rest in bed should be carried out for from three to six months; and if after this time there is some improvement, the patient may be suspended, and put into a plaster of Paris or a poro-plastic jacket.

In some cases counter-irritation in the shape of the actual cautery has seemed to do good applied over the affected spines, but extreme caution is required owing to the tendency to bed sores. If these form the patient must be put on a water bed.

Tonics as cod-liver oil and the syrups of the phosphate or iodide of iron are of particular use.

In recent years, owing to the labours of MacEwen and Horsley, operative measures for the relief of the pressure have taken their place as the treatment of this disease. The cases most suitable are those in which the cord is compressed by inflammatory products, and by removing the spines and laminæ of the affected vertebræ and clearing these products away in many cases very great improvement has resulted. The question is, When is the right time to operate? As a general rule, it is best to try the effect of rest and extension, with tonics for three to six months; and if there is no alteration in the symptoms, an operation should be advised. Cases do recover completely with rest, and this should first be given a trial; and on the other hand, operation could hardly be expected to do much good if extensive myelitis has occurred from the pressure. In cervical cases where the symptoms come on rapidly and involve the respiratory muscles an operation should be done at once.

FRACTURE OF THE SPINE.

This occurs as the result of injury from a blow or a fall on to the spine. The commonest places are in the cervical region and the upper lumbar. The bodies or only the neural arches are fractured, and the spinal cord is compressed by the fragments, and hæmorrhage is frequently found outside the membranes.

Symptoms.—As fracture of the spine comes in the domain of surgery, it will only be described here so far as the nervous system is affected.

The general symptoms are those of shock, attended by vomiting and unconsciousness, from which the patient may never rally.

The local symptoms of the injury are prominence or irregularity of the vertebral spines, and pain and tenderness radiating along the compressed roots.

The symptoms occur suddenly and differ according to the seat and extent of the lesion. If the cord is completely crushed or cut across, there is absolute loss of all motion and sensation below the lesion, the sphincters are affected with incontinence or retention of urine, cutaneous reflex action is increased below the lesion and lost at the levels corresponding to the lesion. The knee jerk is lost in complete transverse lesions (Bastian). Priapism is apt to occur.

Generally speaking therefore, there is pain and irritation along the nerve trunks with paralysis and wasting of muscles supplied by them, at the seat of fracture and complete paralysis without wasting below.

In lesions not quite complete in the mid-dorsal region the above symptoms are modified, and sensation may be partially affected up to the level of the distribution of the damaged root and the knee jerks be increased. A lesion in the upper lumbar part of the spine causes pain and paralysis in the legs followed by rapid wasting and electrical changes, and relaxation of the sphincters with loss of the knee jerks. In the cervico-dorsal region the arms are more or less paralysed for motion and sensation, according to the level of the root of the brachial plexus which is involved ; and as the intercostals are paralysed the breathing is diaphragmatic ; whereas in the upper cervical above the fourth cervical vertebra death is instantaneous or occurs very rapidly from paralysis of the phrenics and intercostals.

In fractures involving the brachial or lumbar enlargement the paralysed and wasted muscles, and the area of anæsthesia will correspond in the arm or leg to the distribution of the particular roots involved, in addition to the affected muscles and skin supplied by the cord below.

Lesions of the cauda equina occur from fractures below the first lumbar vertebra, and present symptoms hardly to be distinguished from those of the lowest end of the cord; but in favour of the former is the escape of the sartorius muscle and the flexors of the hip. The loss of the superficial reflexes corresponding to the level of the lesion is of much diagnostic use.

Prognosis is necessarily grave, especially as myelitis secondary to the primary injury may supervene; but cases often improve up to a certain point and remain stationary if the cord is not severely injured, though, when the cord is completely damaged transversely, death occurs from bed sores or cystitis if the lesion is not sufficiently high up to cause death by asphyxia.

Treatment.—In all cases, if a definite displacement can be made out, and if the patient can stand it, an operation should be performed as early after the accident as possible; for although, if the cord be completely damaged, one cannot hope for any improvement, an operation still gives the patient the best chance for recovery if the lesion is not complete, and at present it is difficult to be absolutely certain about the condition of the cord. Operations undertaken six months after the injury have caused improvement where the cord was not completely cut across.

TUMOURS OF THE SPINE.

The spinal cord is liable to be compressed by growths from outside, which may spring (1) from the bones of the spinal column, or (2) may extend into the spinal canal from without. Of the former are sarcomata and carcinomata, and of the latter aortic aneurysm.

Symptoms.—In some cases a tumour can be felt, and whenever a tumour of the spine is suspected a careful examination of the abdomen should be made for the presence of a growth in front of the spine; but the chief symptoms are due to the spinal nerves, which are inflamed, compressed, or infiltrated by the growth. As a consequence severe, sharp, localised pain, radiating along the spinal nerves, and increased very much by movement, is the most important symptom. Later on hyperæsthesia of the skin supplied by these nerves ensues.

The spinal cord is liable to be compressed without the dura mater becoming involved, and either symptoms of compression occur, or those of acute myelitis from inflammation of the cord. In the former case slow paralysis comes on in the limbs and trunk below the lesion; while in myelitis the paralysis is a matter of a few hours or days, and at the same time sensation may be affected as well as the sphincters, the knee jerks and skin reflexes below the lesion being usually increased.

Prognosis.—The disease as a rule progresses, and lasts a few months in the case of rapid-growing tumours, or years in slow growths.

Treatment.—If possible the growth should be removed, and failing that procedure, palliatives as morphia are required.

COMPRESSION OF THE CORD.

Causes.—(1) In the spinal column, caries, fractures and dislocations, and tumours; (2) in the membranes, tumours and thickening.

Symptoms.—As was seen in the case of tumours of the spinal column, the chief symptoms are due to pressure on the nerve roots and on the spinal cord itself, and though one side may be affected first the other soon follows. Compression of the nerve roots gives rise to pain localised to the particular roots involved, with hyperæsthesia of the skin supplied by them. This is followed by paralysis and atrophy of the muscles and anæsthesia of the skin supplied by these roots, with loss of electrical reactions. Compression of the spinal cord causes paralysis of motion and sometimes of sensation in the parts of the body and limbs which are supplied by the cord below the lesion. These symptoms come on gradually or suddenly, according as the cause of compression is gradual, as in the case of caries, or sudden in that of fracture ; but even in gradual causes the cord symptoms may be rapid, due to acute myelitis being set up. The sphincters are sometimes affected, with retention or incontinence of urine. The deep reflexes are increased unless the cord is completely cut across, when the knee jerk is lost (Bastian). The knee jerk is also absent when the lumbar enlargement is compressed at the third and fourth lumbar roots. The superficial reflexes are lost at the level of the lesion and increased below it.

Pathology.—The cord is flattened or narrowed at the point of compression. In recent cases the cord is soft in consistency, but in cases of longer duration it is harder owing to fibrous overgrowth. The changes are those of inflammation of the whole transverse thickness of the cord, and are the same as that described under acute and chronic myelitis.

CHAPTER VIII.

INFLAMMATION OF THE SPINAL CORD, OR MYELITIS.

This disease may be *acute, subacute* when the onset is from two weeks to two months, and *chronic* if longer than this. The disease is said to be *transverse* when the whole horizontal thickness of the cord is affected, *diffuse* when the inflammation extends up and down the cord over a larger area, *focal* when it involves a local circumscribed area, and *disseminated* when there are numerous foci; the term *polio-myelitis* is used when only the grey matter is involved.

ACUTE MYELITIS.

Causes.—It occurs especially in males, and in adults rather than in children. Of direct causes the most common are exposure to wet and cold, injuries to the spinal cord, as wounds, bruises, and concussion, and rarely to a strain in trying to prevent a fall. Toxic conditions of the blood are the cause in cases following the acute specific fevers, and those after syphilis and gout may be due either to morbid blood states or to disease of the vessels.

Symptoms.—Acute transverse myelitis begins with general symptoms of shivering, headache, and pyrexia, which on the second and third days may be up to 103° or 104°, and with the special symptoms of numbness and tingling in the feet, and sometimes pain there. These are followed by motor symptoms, a difficulty in walking, owing to loss of power and a feeling of weight in the feet; this loss of power gradually ascends, involves the knees, and then the hips, and increases in severity so that in a few hours the patient is unable to move the limbs at all; at the same time there is progressive loss of sensation. The sphincters are affected from the first, usually with retention of urine and fæces. This constitutes an acute case, but in subacute or chronic cases these symptoms may take days or weeks before reaching their maximum.

In cases in which the myelitis is not completely transverse and of less severity, the patient does not lose all power over the legs, and sensation may be unaffected or only diminished.

The subsequent symptoms vary according to the seat of the lesion.

In a transverse myelitis of the lumbar enlargement a week after the onset there is no power to move the legs, the muscles of which begin to waste and do not respond so readily to faradic electrisation, thereby showing that the trophic centres in the cells of the anterior horns are involved; the muscles become quite flaccid, so that the foot drops down as in cases of neuritis; the knee jerk and plantar reflex are absent; the sphincter vesicæ is paralysed, so that the urine dribbles away as it is formed; there is loss of sensation in the legs, the limit of which corresponds to the skin area supplied by the highest part of the cord affected.

Transverse myelitis of the mid-dorsal region differs from the above in that the paralysed muscles do not waste and their electrical reactions are not altered, and the urine is retained or passed reflexly. The knee jerks become excessive, ankle clonus is present, and the plantar reflex is well obtained; there is anæsthesia of the legs, extending up the spine and over the front of the abdomen to the level of the skin area supplied by the dorsal nerve involved by the myelitis; hyperæsthesia exists at this level, and is well detected by passing a hot sponge along the spine.

In the cervical enlargement the symptoms are the same as in the mid-dorsal, except that the intercostals are paralysed, and certain of the muscles of the upper limb, besides being paralysed, soon waste and give electrical changes. The loss of sensation extends up to the upper part of the thorax, and along the arms it corresponds to the skin areas supplied by the highest level of the brachial enlargement affected.

If the lesion involves the fourth cervical roots, the diaphragm is paralysed, and usually death rapidly ensues.

In determining the position of the lesion in the cord, in addition to the amount of motor paralysis, most important data are furnished by the level of the loss of sensation, by what is known as the girdle pain, and also by the skin reflexes. The level of the relative loss of sensation for touch, temperature, and pain should be carefully mapped out from the spine to the middle line in front. The girdle pain is a feeling of painful constriction round the abdomen, and it occurs in the same place as the hyperæsthesia, *i.e.* just above the highest level of the lesion. The superficial reflexes whose spinal centres are below the seat of disease are increased, and those whose centres are involved in the disease are lost. So that in a myelitis of the mid-dorsal region the epigastric and abdominal reflexes would be lost, while the plantar reflex would be increased.

The form of sensation which is lost differs in individual cases—in some sensation to touch, pain, and temperature is completely lost, in others touch is preserved, and sensation to pain and temperature is lost; and as a rule these two are associated together, while loss of muscular sense usually goes with sensation for touch.

The course and duration of the disease vary according to its intensity and extent, and as the acute symptoms subside the case becomes one of chronic myelitis; in some cases sensation returns in a month or two, but motion may be absent for several months.

Other symptoms occur as the disease persists: the legs become rigid, due to descending sclerosis of the involved lateral columns of the cord, and are affected with spasm of the extensors of the knee, called clasp-knife rigidity, from the condition that when the knees are flexed the stiffness is slight, but when they are extended the limbs become quite rigid like an opened clasp-knife, while in other cases the spasm predominates in the flexors of the knee; the reflexes are much increased, so that the legs can be thrown into violent trembling on testing the knee jerk or ankle clonus, and the slightest touch on the sole of the foot causes the legs to be reflexly drawn up at once.

Trophic changes are liable to ensue in the paralysed parts, and especially where there is any pressure, as over the sacrum and on the heels. Here bullæ containing serous fluid are very readily formed, and are easily converted into bed sores.

Changes in the urine, which are partly trophic and partly due to retention, occur, and it becomes alkaline, and from cystitis which may ensue it may contain pus.

Besides the acute myelitis, which is more or less completely transverse, disseminated myelitis occurs, affecting by separate foci different parts of the cord, and the symptoms will correspond to the different parts of the cord as they are involved.

Pathology.—In its naked-eye appearances the spinal cord is softer and more diffluent than natural, and the vessels are congested, so that in the early stage this condition is called red softening, to distinguish it from a later condition, when the colour is paler, and called yellow softening. When the cord is diffluent all structure may be lost, and only the *débris* left, consisting of blood corpuscles, leucocytes, degenerated swollen axis cylinders, myelin granules, corpora amylacea, and cells of various shapes.

Microscopically in the early stages the chief change is the enormous distension of the vessels whose perivascular sheaths are filled with leucocytes and red blood corpuscles, and which are also seen in the tissues outside the vessels. The cells of the grey matter later on are swollen and granular, they contain globules probably fatty, and their processes shrink and gradually disappear, in addition numerous corpuscles are seen in the grey matter, probably the nuclei of the cells which have disappeared; in the white substance the myelin of the fibres disintegrates, and may disappear forming granules, globules of myelin, and "corpora amylacea," and the axis cylinder is much swollen.

The interstitial connective tissue in the white substance may be primarily affected, or secondarily to the nerve tissues, and becomes increased with the formation of the large spider "cells of Deiters."

In transverse myelitis the inflammation is limited as a rule to a small extent up and down the cord, while in the diffuse it extends over a large area up and down but not completely across; in the disseminated form the foci of inflammation are numerous and all over the cord.

The lateral and posterior columns, if involved in the myelitis, cause later descending and ascending sclerosis.

The cause of the inflammation and its especial frequency in the dorsal cord is unknown, but it has probably to do with the peculiar blood supply of the cord.

In cases which recover or pass on to chronic myelitis there is a fibroid overgrowth of the spinal cord connective tissue. If the nerve cells from which the fibres come are preserved, the nerve fibres may recover even if their axis cylinders are considerably damaged.

Diagnosis.—See p. 186.

The **prognosis** as regards life is worst when the disease is in the cervical enlargement owing to the interference with respiration, and it remains serious until the myelitis has ceased spreading. Later on bed sores or cystitis are unfavourable symptoms.

With regard to recovery of power and sensation, this varies with the degree of paralysis, whether absolute or partial, and whether the cause, *e.g.* pressure, can be removed. The prognosis is unfavourable as long as there is no alteration in the symptoms; but as soon as any improvement begins, usually in sensation, there is reason to hope that in time, even in six months or a year, motion may also return. And though rigidity and increased knee jerk, if due to pressure, do not necessarily preclude recovery, even after several months, the condition of flexor spasm where the heels are drawn up to the nates is very bad.

Treatment consists in complete rest. The patient should lie as much as possible on his face, or on one side or the other, and should be put on a water bed to prevent bed sores. In strong patients leeches, or wet or dry cupping, should be applied along the spine, and be followed by hot fomentations.

With regard to internal treatment, the bowels should be kept open, and the action of the skin and diuresis promoted by liq. am. acet., citrate or acetate of potash, and spirit of nitrous ether. Of special drugs ergotin 2 to 4 grains injected hypodermically has seemed to do good in some cases. If there is a history of syphilis, some cases certainly get well quickly with daily inunction of ung. hydrag. when employed at the onset of a case. Iodide of potassium has not done much good even when the disease is stationary, and better results are obtained from tonics such as arsenic, quinine, and iron.

After the acute stage, the chief difficulty is to prevent cystitis and bed sores. Unless the patient can empty his bladder, and a catheter should be passed occasionally to see if there is any residual urine, the urine should be drawn off by catheter, which should be kept absolutely antiseptic; if the urine becomes alkaline or cystitis is set up, the bladder should be washed out every day with boracic lotion. In one case of mine, which recovered, the patient had a rigor with a very high temperature if the bladder were not washed out twice a day. Incontinence of urine when it dribbles away is difficult to manage. A bed urinal is required, but if this causes irritation or sloughing, absorbent wool, as recommended by Gowers, is useful, changed as often as it becomes saturated.

Bed sores should be prevented by position and ring pads over the parts subject to pressure, and by hardening the skin with spirit and water. If they form, iodide of starch (Marshall) or iodoform covered over with an oakum pad is a useful dressing.

Later on, extension by weights and massage may be required for the contracted joints. Electricity is of no use unless the muscles are wasted, due to a lesion of the brachial or lumbar enlargement.

CHRONIC MYELITIS.

This condition may be secondary to acute myelitis, or may primarily develop slowly without showing active inflammation. Like the acute form, the disease may be focal, diffuse, or disseminated, and may extend across the cord completely or partially.

Causes.—It occurs most in early adult life, and equally in both sexes. Direct causes are injuries to the spine and prolonged and frequent exposure to cold and wet, and constitutional changes due to syphilis, gout, and alcohol. The disease is also liable to be set up by external pressure from disease of the spine, etc.

Symptoms.—In those cases which follow after acute myelitis the symptoms will correspond to those described on p. 123.

The primary form begins insidiously with numbness or pins and needles in the feet and legs, with dull pains in the back, and a feeling of constriction round the trunk at the level of the lesion. Sensibility is only slightly affected at first, and is always less than the motor paralysis. After some weeks or months the legs become weak, with a tendency to drag the feet, although there is no absolute loss of movements. Both legs may be affected simultaneously or one before the other. The sphincters are gradually affected, and later the joints of the legs become rigid with increased knee jerk and ankle clonus. The loss of sensation is rarely absolute, and is often only affected for light touches; as a rule it involves both legs, and extends up the trunk to the level of the skin area supplied by the highest part of the cord involved. If the myelitis attacks the brachial or lumbar enlargement, the muscles of the arms and legs will be gradually paralysed and wasted, with electrical changes and with some loss of sensation.

In some cases the loss of sensation and motion begins in the same leg, in others the sensation is first affected in one leg and motion in the other. The course of the disease is slowly progressive, taking two or three years to affect considerably the patient's powers of walking; but though the disease may become stationary at any point, it does not tend to improve, and the prognosis is therefore unfavourable.

Diagnosis.—See p. 188.

Pathology.—The chief change in the cord is an overgrowth of the interstitial fibrous tissue, which is either primary or occurs secondarily to acute myelitis. The position of the changes in the latter is that of the disease it follows, while in the primary form there may be foci in different parts of the cord. The pia mater is much thickened, as are the tracts of fibrous tissue which pass into the cord from it. The interstitial tissue both in the white columns and in the grey matter is much increased, while the nerve fibres waste, and the nerve cells, at first enlarged, gradually shrink and disappear. The vessels of the affected part are increased, and their walls are thickened. Below the lesion there is often descending sclerosis of the lateral columns, and above it there may be ascending sclerosis in the posterior columns.

Treatment.—In both the primary and the secondary form of the disease, tonics, fresh air, and absence from all worry and anxiety are requisite. Complete rest for a time is necessary, but not for more than a month or so, especially in primary cases.

With regard to internal treatment, tonics as iron and quinine are required; and of special nerve tonics arsenic, mercury (perchloride gr. $\frac{1}{16}$, or red iodide gr. $\frac{1}{30}$), and strychnia are most useful. Iodide of potassium may be given if there is a definite history of syphilis; but it has not proved as much use as might have been expected.

As regards local treatment, hot douching of the spine has been recommended (Brown-Séquard); but the best results are obtained from linear counter-irritation by the actual cautery along the side of the spine over the seat of disease, a fresh application being made every second or third day, and thus the irritation can be kept up several weeks. Mustard plasters and blisters may be used for a similar purpose. If the limbs are rigid, massage and movements of the joints will be required, and electricity in the form of the constant current if the muscles are wasted.

ANTERIOR POLIO-MYELITIS, ATROPHIC SPINAL PARALYSIS, INFANTILE PARALYSIS.

This is an acute inflammatory disease, limited to the cells of the anterior cornua of the spinal cord, and characterised by loss of motor power in the limbs developing very rapidly, and followed in a few weeks by atrophy of the muscles affected.

Causes.—From one of its names, it will seem that this disease is very prevalent in childhood; but it does occur occasionally between ten and twenty years of age, or over that. It is most frequent in children of three years and under, and affects either sex about equally. The frequency of cases has been found to vary with the seasons of the year (Sinkler), and to be most numerous in the hot months—June to September. This is probably due to sudden chill after violent heating, as there is no doubt that getting wet and exposure to cold do produce the disease.

Injuries such as falls on to the back have been met with in some instances just before an attack, and they must be considered as direct causes.

Symptoms.—These may come on in a child when in apparently perfect health. A typical case is that of a child who is feverish, has some headache, vomiting, and sometimes general convulsions. It is put to bed, passes a restless night, and in the morning one, two, or even all four limbs are found to be completely paralysed. The temperature is not very high, but remains raised for four or five days. In some cases the onset is very sudden, the paralysis coming on in a few minutes (these cases are probably due to hæmorrhage or thrombosis of the cord); in others it is attended with pain in the limbs and back. Sometimes the paralysis takes one or two days to develop. Sensation is not affected, and the power over the bladder and rectum is not impaired, except in very rare cases. The knee jerk is lost if the extensor cruris is affected. The loss of power which may affect the whole limb or only certain of its muscles is at its worst at the onset, and it remains stationary for from a few days to six or eight weeks, when improvement in power gradually takes place in certain of the muscles.

Those muscles which do not recover begin to waste and become flaccid at the end of two or three weeks' time, and if tested electrically they give the reactions of degeneration, *i.e.* they do not react to a faradic current, or only to a very strong one, and they show increased irritability to the constant current, and especially to the positive pole (see p. 72). In most cases, but not all, those muscles which lose their faradic excitability and rapidly waste lose also in the course of about six months their excitability to the constant current, and are permanently paralysed; while in the muscles which ultimately recover the reaction to the two currents is diminished, but not completely lost for both.

The distribution of the paralysis is more frequently in the legs than the arms, and it is not as a rule symmetrical. The whole limb may be paralysed, or more commonly certain muscles which correspond very often to the groups of muscles assigned to the different segments of the brachial and lumbar enlargements of the cord (see table at end of book). Occasionally the muscles of the trunk are involved, and rarely those supplied by the motor nuclei in the medulla.

After the stage of improvement of those muscles which are going to recover, the patient is left in a chronic condition with some muscles permanently atrophied and paralysed. It is then that deformities occur, such as talipes, owing to the unopposed action of the healthy muscles or of those which have recovered; but in those cases where both extensors and flexors are paralysed the limb is quite flaccid, like a flail. In children the whole limb may be retarded in growth, and be uniformly smaller than on the healthy side; the paralysed limb is also colder, and the circulation not so active.

Pathology.—In the case described by Drummond in which death occurred seven hours after the onset, the spinal cord at one level was red and softened, and under the microscope showed extreme vascularity, especially in the anterior horns, and also in the anterior white columns and the middle and anterior parts of the posterior horns, the arteries being intensely congested with minute hæmorrhages. The neuroglia was swollen, and in the anterior cornua the changes were most marked, the cells being granular and ill-defined, and their nuclei and processes often absent. In cases of longer duration softening of the cord has been found, with disappearance of the anterior horn cells. In the muscles, the muscular fibres in part or completely disappear, and are replaced by fibrous tissue.

The primary lesion is vascular, in most cases inflammatory, and in some hæmorrhagic, but in the absence of an injury, the cause of the lesion is not known.

The destruction of a cell in the anterior horn is followed by atrophy of the muscle fibril which it supplies; and as the cell is not regenerated, the fibril remains permanently paralysed and atrophied.

In extreme cases probably the whole length of the cord is affected, but especially the brachial and lumbar enlargements, and at certain levels or segments the anterior horn cells are destroyed and the muscles supplied by them will be paralysed and atrophy.

Prognosis.—As regards life, the disease is only fatal where the muscles of respiration are involved or the constitutional symptoms are very severe. As regards recovery, an opinion cannot be given till the paralysis has become stationary. At the end of two weeks by electrical testing it is possible to tell that the muscles which react to faradisation will recover, and those which do not react will either be permanently paralysed or recover imperfectly; in the latter cases, as long as the retention of reaction to the constant current remains, there is some hope that the faradic reaction may return, and with it some power in the muscle.

Treatment.—In the acute stage, the patient should be kept absolutely quiet in bed, and lie on the face or on one side, and hot applications be made to the spine. When the attack is due to cold, diaphoretics as liquor ammoniæ acetatis and citrate of potash may be given; ergot and belladonna are also recommended. When the disease becomes stationary, cod-liver oil and tonics as the syrup of the phosphate of iron and quinine are required. Strychnia by the mouth is recommended by

Gowers to be tried from two to four weeks after the disease has become stationary.

With regard to local treatment, it should not be used till at least a month after the onset; the limb should then be bathed every morning in hot salt and water and rubbed with a rough towel, and particular care should be taken to keep the limb warm with thick stockings; the muscles should also be systematically rubbed very night and morning. The question of electricity is very important. The constant current should be employed, the positive pole being applied to the spine with a large thick pad—say 4 × 2 inches—well wetted with salt and hot water, and the affected muscles should be brushed up and down with the negative pole applied with an electrode two inches in diameter; the electrode should be taken off and reapplied at the end of each stroke, so as to make and break the current, the strength of which should be only just sufficient to produce a slight contraction in the muscle. The application should be made for a quarter of an hour every day.

With children it is best to begin without any current at all, and then proceed in the course of a week increasing every day by one or two cells till the proper strength is reached, or as much as the child can bear. In some children the application produces so much fright and general disturbance that it is best to discontinue it.

Though electricity has probably no effect on restoring the functions of the damaged cells in the anterior horns of the spinal cord, it is beneficial in keeping up the excitability of the muscles, so that should the cells recover, the muscles will be in the best condition to respond to impulses emanating from these cells. As long as the muscles respond to the constant current, treatment may be continued; when this fails, it is useless.

The patient should be encouraged to try and move the paralysed muscles, and definite directions should be given to the friends to see that an attempt should be made every day to perform some prescribed movement, which at first should be passively assisted.

Deformities caused by overaction of unaffected muscles must be counteracted by supporting the foot in the proper position by sand-bags; and when there is no hope of the muscles regaining power, tenotomy of the contracted muscles and surgical boots may be necessary to enable the patient to walk.

ACUTE ASCENDING PARALYSIS.

This disease (also called Landry's paralysis, by whom it was first described) occurs very rarely. It is characterised by paralysis, beginning in the legs, and gradually in a few days involving the trunk, thorax, arms, diaphragm, neck, and pharynx. It often ends fatally, and on examination nothing pathological has been found to account for the symptoms.

Causes.—The disease has occurred after severe chills and exposure to cold, after febrile diseases as typhoid fever, or after slight wounds.

Symptoms.—Weakness in both legs is usually the first symptom preceded sometimes by general malaise, the thighs are soon affected, and in a few hours the patient is unable to walk; the muscles of the pelvis and abdomen, and then those of the thorax, are involved; later the upper limbs are paralysed, beginning at the shoulder or in the hands. The muscles of the neck and diaphragm next suffer, and if completely paralysed death ensues; in other less severe cases the muscles of swallowing and the soft palate are affected with the lips.

As a rule the muscles of the limbs and trunk are completely paralysed and flaccid, but they do not waste, and their electrical reactions are not altered. Sensation is not affected. The cutaneous reflexes are abolished, and the knee jerk is lost. The sphincters are not affected, and as a rule there is no pyrexia. The spleen is enlarged (Westphal).

The duration of the disease is from two days to a week, and cases which do not die in that time may remain several weeks paralysed, and then take several weeks more to recover. The order of the muscles involved may vary from that given above.

Pathology.—As a rule nothing has been found post-mortem, but recently changes have been described in the peripheral nerves (Eisenlohr). It seems possible that the disease may be due to some toxic condition of the blood, similar to diphtheritic paralysis or the form of paralytic rabies seen in rabbits.

Diagnosis has to be made from acute peripheral neuritis, diphtheritic paralysis, acute polio-myelitis, and acute myelitis; it is distinguished by the rapid ascending paralysis, with absence of pyrexia, of pain, and of affection of the sphincters, and absence of wasting and electrical changes in the muscles.

Prognosis is very bad, and is more serious in proportion as the respiratory muscles are affected. The majority of cases end fatally.

Treatment.—Complete rest, with hot-air bath and diaphoretics, followed by counter-irritation to the spine, should be employed. Ergotin gr. i to iij given every hour has been recommended by Gowers, and also iodide of mercury. Nasal tube feeding may be required.

DIVERS' PARALYSIS.

This form of paralysis occurs in those who, after working under high pressure in caissons or in a diver's dress, come too suddenly into the ordinary atmospheric pressure.

A pressure of two atmospheres, or a depth of water from fifty to a hundred feet, and prolonged working seem to be requisite to produce the paralysis, which does not come on till the surface is reached, when it may occur immediately or within an hour. The onset is usually sudden with weakness, followed by paralysis of both legs or rarely the arms, and with loss of sensation and affection of the sphincters. The paralysis may be slight and recovered from in a few days, or severe and last several weeks. Rarely the paralysis is hemiplegic, with cerebral symptoms, as coma.

The symptoms are like those of myelitis, only more rapid in their onset, and may be explained by the theory that the increased pressure causes increased absorption of air by the blood in the lungs, and the pressure being taken off rather suddenly the spinal cord is damaged by the sudden escaping of the compressed air into the subarachnoid space. One patient under my care was a pearl-diver, and some months after the onset had all the symptoms of secondary chronic myelitis. In one case of Leyden's, fissures were found in the cord. The treatment is that of acute myelitis.

HÆMORRHAGE INTO THE SPINAL CORD.

This condition, which is very rare as a primary condition, occurs much less frequently than was formerly supposed. It occurs more often in the earlier half of adult life than in the latter, and is usually secondary to an injury to the spine or to a commencing myelitis; the blood is sometimes extravasated into tumours and cavities of the cord. The hæmorrhage is met with in the grey matter as a rule, as minute extravasations, and when secondary to myelitis this latter is found to involve the cord outside the area of hæmorrhage.

The especial symptom which separates hæmorrhage from other diseases of the cord is the sudden onset, with pain in the back, with almost instant paralysis of the limbs, usually the lower, for motion and sometimes also for sensation, with affection of the sphincters. The muscles are generally flaccid when paralysed, and the knee jerk may be at first abolished from shock, but soon becomes increased if the lesion be above the lumbar enlargement. The symptoms may show subsequent extension of myelitis up or down the cord.

The prognosis is serious until the symptoms become stationary, and then it depends on the amount of the subsequent myelitis.

The immediate treatment consists in complete rest in the prone position, ice to the spine, and the hypodermic injection of gr. ij to iij of ergotin; and subsequently the treatment is that required for myelitis.

CHAPTER IX.

DEGENERATION OF THE SPINAL CORD.

(1) OF THE WHITE MATTER.

Primary Spastic Paralysis.

This is characterised by a primary rigidity of the legs, coming on gradually with increase of all the deep reflexes, and with slight weakness in the legs. It is also called primary lateral sclerosis.

Causes are exposure to wet and cold and extreme over-fatigue, and the disease sometimes follows syphilis. A neurotic family history occurs occasionally. Between twenty and forty is the most common period of life, and the two sexes are about equally affected.

Symptoms begin gradually first in one leg and then in the other with a feeling of heaviness and stiffness followed by some weakness. On examination it is found that all the movements of the legs can be performed, and often with normal strength, or there is perhaps some difficulty in dorso-flexing the ankles. The most prominent feature is the rigidity of the legs, which, although relaxed when flexed at the knee, become when extended absolutely rigid—a condition called "clasp-knife rigidity,"—and cannot then be flexed passively till the spasm becomes relaxed. This rigidity produces the characteristic spastic walk, in which the feet are pulled along the ground as if they were glued to it, and the patient " hitches up " the shoulders alternately to diminish the friction of the foot along the ground. The rigidity is usually not continuous, but is readily produced on passively moving the limb. All the so-called tendon reflexes are excessive, the knee jerk, rectus clonus, ankle clonus, wrist tap being readily obtained, and even in some cases the muscles of the lower jaw respond to a tap on the chin, giving the jaw jerk. Both in walking and while sitting ankle clonus is spontaneously produced causing a rhythmical tremor of the legs. In well-marked cases the whole limb at times passes into a general clonus.

Cutaneous reflex action is usually increased and readily brings on a spasm. The muscles are well developed and have normal electric reactions, the sphincters are not as a rule affected, and sensation is normal ; dull pains and pins and needles are sometimes complained of in the legs and back. Later on the arms may be affected like the legs, with rigidity and some weakness, and rarely spasm of the back muscles occurs.

Of other symptoms, nystagmus of the eyes should be looked for, as it is found in some cases, and is of importance as a symptom of disseminated sclerosis, a disease which occasionally begins with lateral sclerosis. The pupils and optic discs are normal. The sphincters are sometimes involved, and sexual power may be lost.

Pathology.—The hypothetical assumption is that the lesion is a degeneration or sclerosis of both lateral columns beginning below in the lumbar region and subsequently passing up the cord, but without involving the cells in the anterior horns of the cord, though at present the question has not been satisfactorily settled by autopsy.

Diagnosis.—See p. 188.

Prognosis is not favourable to recovery, but is not dangerous as regards life; and although the progress is often very slow, and lasts for years, the disease does become stationary; it is however hardly possible to expect improvement when the legs are markedly spastic.

Treatment.—Nerve tonics such as arsenic are recommended, and for the spasmodic condition of the legs belladonna and cannabis Indica with the bromides should be tried. Warm baths prolonged for an hour, and the constant current applied as the galvanic bath or with the anode to the neck and the feet in water connected with the cathode, seem sometimes to diminish the spasm. Strychnia and faradisation of the muscles increase it.

Complete rest, with systematic massage of the muscles and Turkish bath (Gowers), has done good in relieving the spasm.

As the legs are frequently drawn up spasmodically as the patient is going to sleep, an extension apparatus of weights passing over pulleys and attached to each foot is useful to counteract this movement.

Locomotor Ataxia.

This common disease (which is also called tabes dorsalis) is characterised by symptoms of inco-ordination, of sharp pains and loss of sensation in the limbs, but without as a rule any loss of power in them.

Causes.—Hereditary nervous disease has not much influence. The disease is not common before thirty, and is most frequent between this and forty, and it may begin later, even up to sixty. Men are very much more often attacked than women. Syphilis, as first shown by Fournier, occurs as a very important predisposing cause in from one-half to two-thirds of the cases, and it may be acquired or inherited, locomotor ataxia coming on from five to ten years after the acquired form. Of other causes, injuries to the back and exposure to wet and cold appear to be predisposing.

Symptoms.—The onset is gradual.

Motor symptoms.—Except in rare instances, and in the later stages, there is no actual loss of power or of movement in the limbs, the patient when sitting down being able to execute movements with normal strength. The ocular muscles may be paralysed as a transient symptom in the early stages or permanently in the later stages of the disease. In the early stages the external rectus is frequently paralysed, while later the whole third nerve of one side, or combined paralyses of associated movements of the two eyes, occurs. Inco-ordination of movements is one of the most important symptoms, and one of its earliest signs is a sudden giving way of the knees as the person is walking or standing (Buzzard); later on there is difficulty in standing with the eyes shut when the bare feet are put close together, the patient swaying about or falling to one side or forwards—a symptom often discovered by the patient in closing the eyes to wash the face. In trying this test it is important to ascertain that the patient is standing steadily with the eyes open, before noting the effect of closing them. Inco-ordination is also tested by making the patient, when lying down with the eyes shut, touch the knee of one leg with the heel of the other. Incoordination of the upper limbs occurs usually late in the disease, and is found out by making the patient touch the tip of his nose with the eyes open and shut; in the latter condition the finger is landed on the cheek or chin instead of on the nose.

The gait when present is typical, and consists in the patient throwing out the legs in an irregular manner, lifting the feet too high, the heel being brought down with a stamp with the toes turned upwards, while the feet are kept far apart, and in turning round there is considerable staggering. The difficulty is increased if the patient does not look at his feet, and is due rather to an inability to make the legs go where they are required, than to a loss of equilibrium of the whole body. In the early stages slight difficulty of walking can be found out by making the patient walk barefooted, toe and heel, along a straight line. Unusual forms of paralysis are those of the muscles of mastication of one side, and the abductors of the vocal cords, which may be bilaterally affected, so that the cords come together in inspiration instead of separating.

The knee jerk is lost early in the disease, and usually one side before the other, and it cannot be obtained even by reinforcement (see p. 74). In very recent cases, it is found on one or both sides, and disappears under observation.

The reflex action of the pupil to light is lost early in a large proportion of cases and in both eyes as a rule, while contraction on accommodation is retained, forming the Argyll-Robertson symptom; and also the dilation which is normally observed on pricking the skin of the neck is usually lost (Erb). The pupils are generally contracted, and are often unequal. In some cases they are "pin-point" in size, but they may be normal in size, and especially in young patients.

The superficial plantar reflex is present at first, but is subsequently lost.

The organic reflexes are often affected early, so that the patient has difficulty in passing or in holding his water, or he never completely empties his bladder, a few ounces being retained, which may easily become alkaline and set up cystitis ; also he is unable to retain his motions, which are passed involuntarily. Constipation is frequently present.

Sexual power is frequently lost early in the disease.

Sensory symptoms.—One of the earliest symptoms is the characteristic *pains*, which are very sharp, boring, burning, or cutting in character, and consist of a series of stabs or darts, each lasting a second or so, and are so sudden that they are gone before the patient has time to cry out. They are often considered by the patient to be "rheumatics," and to have no connection with the other symptoms of tabes. These sharp pains may be preceded by dull aching pains in the limbs, but these latter are not diagnostic of the disease. Occasionally the pains are of a more continuous character, and are described as burning or gnawing, but they are not so characteristic as the sharp pains. The pains may occur months or years before any other symptom, and are met with especially in the legs, shifting their position about the knee, ankle, and calf, but not following the course of the peripheral nerves. They occur sometimes in the arms, especially along the ulnar border, and rarely in the head. Around the trunk, pains of a continuous tight band-like nature occur, called cuirass pains. The pains are always worse in cold damp weather.

Subjective sensations of a feeling of tingling, creeping, are complained of in the limbs, or one leg may feel ice-cold and the other burning hot to the patient.

Sensibility is frequently diminished or lost in the soles of the feet at first, and differently for the various forms of sensation; the most common being a loss for painful and temperature impressions, which are felt as touches, and not for tactile impressions; or tactile sensations may be lost or incorrectly localised, while the feeling of pressure is retained. A delay in appreciating stimuli—especially painful ones—occurs for from one to five seconds or more. The distribution of the loss of sensation in the legs is sometimes in patches, in others it corresponds to the root distribution. It may extend up the leg and involve the trunk up to the second intercostal space. In the arms loss of sensation begins usually in the third and fourth fingers and on the ulnar side of the forearm. Loss of sensation for temperature usually coincides in area with that for painful impressions.

Loss of muscular sense—*i.e.* the loss of the recognition of the position in which a joint is placed when the eyes are closed—frequently accompanies the loss of tactile sensation, and in many cases the sensibility of the muscles and joints to passive movement is lost.

Special senses.—Loss of *sight*, which may become absolute, from a primary optic atrophy, is rather a common symptom, and when present occurs early in the disease often as the first symptom; it is usually preceded by a peripheral diminution of the field of vision, and it progresses gradually, affecting both eyes and one often before the other. The discs present to the ophthalmoscope the grey appearance of a progressive atrophy without much alteration in the size of the vessels and without filling up of the central cup. Loss of hearing to aerial and perosseal sounds may also occur very rarely.

Trophic changes occur as an early symptom in the joints, and the condition is named after Charcot, who first drew attention to them (tabetic arthropathy, Charcot's joint). The first change seems to occur in the ligaments, which become lax, perhaps from sudden strains or blows, so that the knee can be over-extended; then effusion takes place into the joint, and is followed by erosion of the cartilage and atrophy of the ends of the bones, while occasionally irregular bony outgrowths are met with. The condition is usually quite painless, and affects the large joints, especially the knee, hip, and shoulder, and occasionally the elbow and toes. A form of "club-foot" also occurs. Sudden fracture of the long bones due to trophic changes follows from slight causes, as turning over in bed.

Perforating ulcer of the foot, which begins as a hard corn and resists all treatment, should always suggest an examination for other symptoms of locomotor ataxia, as it is in this disease that this condition is most often met with as a trophic lesion.

Other trophic changes are herpes of the skin, changes in the nails, and painless dropping out of the teeth.

Visceral symptoms which most commonly occur are gastric crises,—attacks lasting for a whole day or longer of most severe epigastric pain, attended by incessant purposeless vomiting quite independent of food; laryngeal crises, or attacks of spasm of the larynx, with dyspnœa and much stridor; and rectal crises attended with tenesmus. Some patients have unpleasant visceral subjective symptoms as though the intestines were being wound up.

Three stages of locomotor ataxia are usually described which come on gradually, and as a rule progressively.

(1) The præ-ataxic stage, in which the symptoms are sharp pains, loss of the knee jerk, slight unsteadiness in standing after closing the eyes, non-reaction of the pupils to light, slight anæsthesia in the feet, and some difficulty of controlling the bladder, may last from a year or two up to twenty years.

(2) The ataxic stage, in which walking is only just possible with the aid of help, and where the above symptoms persist, or become more marked.

(3) This, which is sometimes called the paralytic stage but which is an extreme condition of the second stage, is characterised by inability of the patient to walk or stand, and may last ten or twenty years. There is usually only slight wasting and weakness of the muscles of the limbs; but the inco-ordination is very pronounced, the legs being thrown about very violently on any attempt to move them while lying down, and the loss of sensation is extreme. The reflex action of the bladder is lost, and the urine has to be drawn off.

One form of the disease begins with double optic atrophy followed by loss of the knee jerk, slight loss of

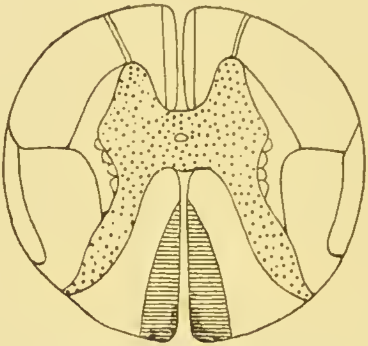

Fig. 21.

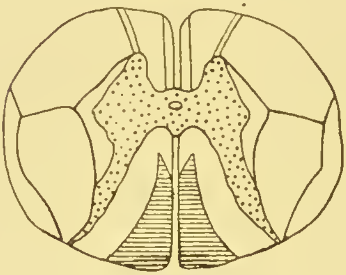

Fig. 22.

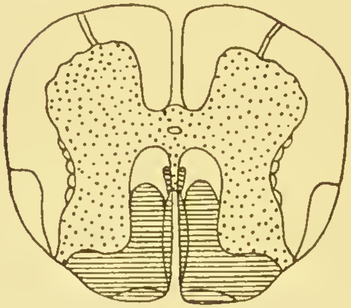

Fig. 23.

Figs. 21, 22, 23 are diagrams of transverse sections of the spinal cord in the cervical, dorsal, and lumbar regions respectively. In fig. 23 the whole of the posterior columns is sclerosed, while in figs. 21 and 22 only the posterior median columns are affected.

sensation and some pains in the feet or in the hands, loss of the light reaction of the pupil, but no inco-ordination of the lower limbs, so that the patient is able to walk if led. In one of my cases optic atrophy had lasted ten years before the patient complained of numbness in the hands, which has now progressed to marked anæsthesia and inco-ordination in the arms while neither of these symptoms are present in the legs, the lesion being probably confined to the cervical part of the cord.

Pathology.—The disease consists essentially of a degeneration in the *white matter* of the nerve fibres and of a sclerosis or overgrowth of the connective tissue of the posterior columns of the spinal cord, beginning in the *lumbar* region in the postero-external columns (Burdach's), and especially involving the posterior roots of the spinal nerves, leaving the part next to the posterior commissure intact (fig. 23). In slight cases secondary ascending degenerations occur in the posterior median columns (Goll's) of the *dorsal* region, and sometimes besides this a narrow band of sclerosis is seen just internal to the posterior cornu, and what is known as the comma-shaped tract situated towards the anterior part of the posterior external column is sclerosed; in the cervical region only the postero-median column is degenerated. In severe cases, however, the whole of the posterior columns along their full extent may be sclerosed. On the other hand in cases, which begin with symptoms in the upper limbs, the spinal cord in the cervical enlargement is sclerosed in the postero-external columns, especially in their outer and hinder parts.

The changes consist of (1) a diminution or absence of nerve fibres, and (2) an overgrowth of the connective tissue of the cord, especially of that forming the septa and trabeculæ, and this is very marked around the blood vessels whose walls are thickened.

In the *grey matter* the posterior vesicular columns of Clarke are degenerated, and very rarely the motor cells in the anterior horns.

In marked cases degeneration has been found in the posterior roots of the cord as far as the posterior root ganglia, and in other cases in the peripheral ends of the sensory nerves.

The optic nerves atrophy, with wasting of the nerve fibres, and with overgrowth of connective tissue.

From the fact that the lesion is confined to certain tracts or "systems" of the cord, and that the peripheral nerves are affected in some case as well as the cords, it seems probable that the disease begins primarily in the nerve elements themselves, and secondarily the sclerosis occurs.

The neuron of the cells in the posterior root ganglion has been considered by some as the starting-point of the disease, and the prolongations of the neuron in the posterior root and the posterior columns degenerate in consequence.

Although syphilis is such a frequent predisposing cause, the lesion is not syphilitic in type but rather a degeneration occurring as a sequel to the specific poison; this would explain the failure of anti-syphilitic treatment in many cases of tabes.

With regard to the symptoms, the pains, anæsthesia and loss of the knee jerk would be produced by degeneration of the posterior roots, and might also perhaps account for the inco-ordination—by cutting off the afferent impressions from the muscles as well as from the skin—and for the trophic changes in the joints. Laryngeal and gastric crises are due to degeneration of the vagus or its accessory nucleus.

The non-reaction of the pupil to light is probably due to a degeneration in the path between the optic centres and the third nerve nucleus.

Diagnosis.—See p. 191.

Prognosis is most favourable in the early cases, and the sooner a case is put under treatment the better. I have seen cases, which were hardly able to walk with help, improve so as to walk alone, and others which have remained stationary for several years without progressing, so that the patient was able to follow his work. Other cases do badly and progress quickly, and it is not possible to tell, except by the rapidity with which the symptoms come on, that the case is unfavourable. The great thing is not to tell a patient, seen in the early stages, that he has a hopelessly incurable progressive disease, as is sometimes done, for cases do improve sufficiently to enable patients to carry on their occupation for years. Cases which end fatally die from pyæmia or renal disease secondary to cystitis, or from exhaustion in the third stage of the disease. Bilateral abductor paralysis of the larynx might easily cause death, if the condition were not recognised.

Treatment.—With regard to general conditions, it is most essential that the patient should be relieved as much as possible from all worry and anxiety, and also from occupation which exposes him to wet and cold; excessive mental work is also harmful if it produces fatigue. The patient should lead a very regular life, and all excesses, whether alcoholic or sexual, must be interdicted; luckily the patient is often saved from the latter by becoming impotent.

In the early stages, especially when the symptoms are stationary, a sea voyage is often beneficial, or a complete holiday away from all work for from three to six months at the seaside.

With regard to medicines, where there is a history of syphilis a course of the liq. hydrarg. perchlor. ʒj ter die for two and increased months, followed by a course of iodide of potassium gr. v to xx, certainly seems to do good in some cases, but not in all. Other medicines which give the best results are arsenic, strychnine, quinine, and belladonna. The first two may be given in the form of a pill containing gr. $\frac{1}{60}$ to $\frac{1}{30}$ of each. Nitrate of silver was formerly much given in doses of gr. $\frac{1}{6}$ to $\frac{1}{3}$. Chloride of aluminium gr. ij to iv twice or thrice a day is recommended by Gowers. I have seen one case improve very much after taking the double chloride of gold and sodium gr. $\frac{1}{12}$ to $\frac{1}{8}$ three times a day.

For symptomatic treatment the pains are those which give the most trouble; antipyrin, antifebrin, exalgin (℥ss. to ℥j of the mistura, or gr. ij in pill), phenacetin gr. iij, cannabis Indica gr. $\frac{1}{3}$ of the extract three or four times a day, are most useful. Morphia and cocaine hypodermically should not be used except in extreme cases of pain, such as gastric crises, and only when other remedies have failed, owing to the ease with which the "habit" is developed. Local applications are not of much use in controlling the pains, but occasionally chloroform poured on to lint and applied to the painful part till marked burning sensation is produced is useful.

When there is difficulty with the bladder, and if on passing a catheter after the patient has finished micturition some urine can still be drawn off, the patient must pass a catheter every day or other day; and if the urine is alkaline, he should wash out the bladder with a weak solution of boracic acid. The patient can do this by himself, if it is strongly pointed out that he must keep the catheter aseptic. Charcot recommends ergot in cases where the bladder is affected.

Trophic changes of the joints and perforating ulcer require absolute rest, and the affected joints should be kept fixed by plaster of Paris, leather, or poro-plastic splints.

Electrical treatment in the form of the constant current has been much used abroad, but it has not found much favour in England. It is applied with the negative pole behind the ear, and the positive pole is moved up and down over the lumbar spines. Tepid baths of about 80° F. with gentle rubbing of the skin and wet compresses on the abdomen at night are recommended (Strümpell). Nerve stretching and suspension of the patient are not to be advised.

Ataxic Paraplegia.

This disease is characterised by some of the symptoms of lateral sclerosis and by some of posterior sclerosis, and is sometimes called combined lateral and posterior sclerosis. The symptoms are weakness and rigidity of the lower limbs, and increased knee jerks with ataxia.

Causes.—Age is usually between thirty and fifty, and males are affected more than females. Exposure to wet and cold is sometimes a cause, but syphilis is much less frequent than in tabes. Sexual excess has been noted before some cases.

Symptoms.—The onset is gradual, with a dull pain in the back and a feeling of weakness in the legs and unsteadiness in standing with the eyes closed ; later the walk becomes affected, the patient has some difficulty in clearing the ground with his feet, while at the same time he is unsteady, especially on turning round, so that the gait is a mixture of that seen in lateral sclerosis and in locomotor ataxia.

In the early stages there is weakness, but no wasting and no definite paralysis of the muscles of the legs, the joints are stiff owing to the spasm of the muscles, the knee jerks are excessive, and sometimes ankle clonus is present. In some cases the inco-ordination affects the upper limbs and with increase of the deep reflexes. The sphincters may be affected with difficulty of holding or passing water. Sexual power is often lost. The pupils as a rule react to light and accommodation. Sensory symptoms, such as lightning pains and anæsthesia, are usually absent. Optic atrophy is rare. Occasional symptoms are a thickness of articulation and nystagmus on lateral movement of the eyes. Later on the weakness develops into definite paralysis of the legs with increased rigidity, but without wasting of the muscles.

Pathology consists of sclerosis of the lateral columns and of part of the posterior columns ; the latter are usually not so much affected in the lumbar as in the dorsal cord, and in the lumbar the posterior root zone next to the posterior cornu is not involved by the sclerosis. This escape of the posterior root zone is the opposite to what is found in locomotor ataxia, and would account for the knee jerk not being lost, while in the dorsal cord the sclerosis of the posterior columns would explain the inco-ordination. The lateral column sclerosis would account for the rigidity, the weakness, and the increased deep reflexes of the limbs.

Diagnosis has to be made in the early stages from locomotor ataxia; later on from primary lateral sclerosis, in which disease there is no ataxia; and from hereditary ataxia, where the knee jerk is absent. Cerebellar disease is distinguished from ataxic paraplegia by the inco-ordination not being increased when the eyes are closed in the former disease. The later stages of the disease are very like disseminated sclerosis, so that it is sometimes impossible to separate them.

Prognosis and **treatment** are the same as primary lateral sclerosis.

HEREDITARY ATAXIA.

This malady (also called Friedreich's disease, from the physician who first described it) is characterised by occurring in several children of a family, and by the symptoms of inco-ordination, absence of the knee jerk, nystagmus, thick speech, with lateral curvature of the spine and deformity of the feet.

Causes.—*Heredity.* Though it is rare to be able to find the actual disease in the previous generation, a neurotic history of epilepsy, insanity, or alcoholism is often present. The disease attacks several of the children in a family, and the males perhaps more than the females.

Age.—In some cases the child is said never to have walked properly, in others at about seven years the first symptoms come on, while in others not till puberty.

Symptoms begin with unsteadiness in walking and liability to fall down ; this is much increased by closing the eyes, but there is as a rule no history of shooting pains. If examined now, besides the ataxia the knee jerk is found to be absent, sensation is as a rule normal, and the pupils react to light and accommodation. Later on there is nystagmus, which is conjugate, *i.e.* the jerking movements of the two eyes are parallel and in the same direction, and it is seen only on extreme movement laterally or upwards of the eyes ; also tremor of the head and neck is observed on voluntary movement together with a peculiar speech in which the syllables are run together. Later still the inco-ordination is so marked that the patient cannot walk or stand alone even with the eyes open, and there is also marked inco-ordination of the upper limbs with the eyes closed. At first there is no paralysis and no wasting of the muscles, but later in the disease there is weakness of the trunk and the leg muscles, so that lateral curvature of the spine is observed, and the feet assume the Z or zigzag position, in which the side of the foot is arched concavely, the metatarso-phalangeal joints are hyper-extended, and the phalangeal joints are flexed (this is due probably to wasting of the interossei), and there is difficulty in dorso-flexing the ankles, with electrical changes in the anterior tibial muscles.

Sensation as a rule is not affected, though there may be dull pains and slight anæsthesia. The sphincters are not affected, and there are no joint changes as in tabes dorsalis.

The course of the disease is very variable, and though it does not improve it may become stationary, and it does not as a rule shorten life.

Pathology.—There is sclerosis of the lateral columns, and also of the posterior columns, and the lesion differs from ataxic paraplegia in being much more extensive, and it involves the posterior root zone in all regions of the cord, and the whole of the posterior columns except the small region to the inner side of the neck of the posterior cornu. The direct cerebellar tract is also involved in the lateral sclerosis, which extends outwards to the surface of the cord and forwards along its periphery. The anterior horn cells are as a rule healthy. The loss of the knee jerk is due to the posterior roots being involved. There is probably a congenital tendency of the nerve tissues to degenerate early, which would explain the occurrence of this disease in such young subjects. Most authors consider that the degeneration of the fibres is the primary change and the overgrowth of connective tissue forming the sclerosis is secondary.

Treatment is similar to that for locomotor ataxia; but it has not much tendency to alter the course of the disease.

(2) DEGENERATIONS OF THE GREY MATTER.

Chronic Spinal Muscular Atrophy.

Under this heading will be described three forms of disease: (1) progressive muscular atrophy; (2) chronic bulbar paralysis;[1] (3) amyotrophic lateral sclerosis.

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[1] Bulbar paralysis is really an intra-cranial disease, but it is more convenient to describe it here.

In all three the etiology is similar.

Causes.—*Age.* The symptoms usually begin between thirty and forty, but these limits may be exceeded. *Sex*—males are affected more than females. A hereditary neurotic history can be traced in many cases, but rarely a direct transmission of the disease. As exciting causes, exposure to wet and cold, anxiety, worry, and in some cases falls on to the arm or back, seem to have started the disease. Although some cases come on after syphilis, there does not seem to be sufficient grounds for looking on syphilis as a cause.

Symptoms.—It will be advisable to take each of the three forms separately :—

(1) *Progressive muscular atrophy* is a slow, gradual wasting of the muscles, and as they waste there is a corresponding weakness and a difficulty of movement. The disease almost always begins in the muscles of the hand, and is symmetrical, one side being a few months in advance of the other. The ball of the thumb is first affected, the small muscles of the thumb waste and become weak so that the thumb cannot be abducted from the first finger, and after them the interossei, lumbricales, and the small muscles of the little finger are involved, so that the fingers cannot be separated; the palm is flattened, and the flexor tendons become visible there; the dorsal interosseous spaces are very apparent, and no muscle is felt on the dorsal and radial side of the first metacarpal bone on the patient pressing the thumb against the index-finger; the flexors, and then the extensors, of the fingers and wrist are affected, and later the supinator longus, biceps and deltoid, and the supra- and infra-spinati. As a rule

the triceps, latissimus dorsi, and the lower half of the pectoralis major are the last to be affected. Of the scapular muscles the serratus magnus and the trapezius (except the highest part) are liable to be wasted. In some cases the cervical spinal muscles are involved, so that the neck cannot be extended, and the head falls forwards with the chin on the sternum. The erector spinæ may be wasted causing cyphosis, and occasionally the sterno-mastoids. The intercostals often suffer, and more rarely the diaphragm ; the legs are frequently not affected, but sometimes the glutei, extensor cruris, and muscles on the front of the leg are involved.

Although the order given above is the most common, some cases begin with wasting of the deltoids spreading to the other muscles of the shoulder, and others with wasting of the anterior tibial muscles.

The wasting of the muscle is by fibrils, and as the muscle wastes so the power diminishes. Fibrillar twitchings or flickerings are often seen in the muscles, and this is well brought out on tapping the muscles, when their irritability to direct percussion will be noted to be increased. The electrical reactions are altered, and are peculiar in that the reaction of the nerves to faradism and galvanism is normal, though the contraction is weak, while to faradism the muscles give a weak contraction to a normal current, and to the constant current the muscles give the reaction of degeneration with an exalted excitability and a slow mode of contraction, responding to the anode (positive) closure before the cathode (negative); later on, when all the fibrils are degenerated, the faradic is abolished, and later still the reaction to the constant current. This is called the partial reaction of degeneration, the muscles contain some normal fibrils which react to the faradic current, but the fibrils which are degenerating react to galvanism sooner than the normal fibrils, and mask their contraction.

The knee jerks, if altered, are increased, except when the quadriceps extensor cruris is involved, when they are lost. The sphincters are not affected.

The wasting of the limbs proceeds till there is little but skin and bone, and with the exception of one or two muscles (as the triceps) all muscle has disappeared, so that the arms hang like flails. There is never any rigidity in the joints. Trophic changes in the skin and joints are not met with.

Sensation is never affected, though dull pains are occasionally felt in the limbs.

(2) *Chronic bulbar paralysis.*—This disease (which is also called glosso-labio-laryngeal paralysis) may occur independently of, or associated with, progressive muscular atrophy. The symptoms are wasting of the muscles of the tongue, so that the mucous membrane is thrown into folds and fibrillar twitchings are visible beneath it, with gradual inability to protrude the organ or to move it in the mouth; wasting and paralysis of the orbicularis oris, producing inability to whistle or to close the mouth, and a peculiar lugubrious expression due to depression of the angles of the mouth and eversion of the lower lip; difficulty of swallowing even of the saliva, which dribbles from the mouth or hangs in festoons about the fauces; paralysis of elevation of the soft palate, and of contraction of the vocal cords. At first the speech is nasal, with loss of the linguals, dentals, and labials, the word "grub" when pronounced becoming

"ngrum," owing to the soft palate being paralysed, and it hangs motionless on attempts at phonation; and as the patient cannot close the posterior nares by the soft palate and prevent air passing through them, he cannot inflate the cheeks unless the mouth and the anterior nares are first closed with the finger and thumb. Speech is eventually lost, only a monotonous grunt being obtained. The movements of the middle and the upper part of the face—including the zygomatici, elevator of upper lip, orbicularis oculi and frontalis—and of the eyeballs are as a rule not affected.

The symptoms are always bilateral, and gradually progress. Swallowing is the great difficulty, as in advanced cases the tongue is almost motionless, and fluids, if they are made to reach the pharynx, are liable to pass up into the posterior nares or down into the glottis. The profuse salivation is a great trouble, owing to the patient being unable to swallow it. The muscles react with diminished force to both currents, or they give the partial reaction of degeneration. Reflex action of the soft palate is lost when this is paralysed. Respiration and cardiac symptoms occur if the vagus nucleus is involved. Sensation is not affected.

(3) *Amyotrophic lateral sclerosis.*—This name was given by Charcot to a condition in which the symptoms of the two above forms are associated with lateral sclerosis of the spinal cord, which occurs either primarily or secondarily. Some authorities (Leyden, Gowers) do not think that there is sufficient evidence for the foregoing symptoms to constitute this a separate disease apart from the two forms described above,

on the grounds that in all cases of progressive muscular atrophy there is sclerosis of the lateral columns; but as there are undoubtedly cases, of which I have had one, where there is no lateral sclerosis, we must admit two separate diseases, progressive muscular atrophy and amyotrophic lateral sclerosis.

Its characters are gradual atrophy of the muscles of the hands, bulbar symptoms, and increase of the so-called tendon reflexes, with some rigidity. When the condition is fully developed, there is in the upper limb wasting of the muscles, as in ordinary muscular atrophy, with an excessive contraction of the elbow flexors on tapping the lower end of the radius, and with some rigidity; there is much rigidity, especially of the legs, which are often not wasted, excessive knee jerks, and ankle clonus; along with the bulbar symptoms there is increase of the jaw jerk, and sometimes a clonus can be obtained by putting the finger on to the incisor teeth and tapping it. In most cases the patient complains of atrophy of the muscles of the thumb, and it is then found that there is some rigidity in all the joints of the upper limbs with increased deep reflexes. Later on, bulbar paralysis similar to that already described gradually comes on, and sometimes involves the muscles of mastication. The legs subsequently become very rigid, without wasting, so as to prevent the patient walking.

In other cases rigidity of all four limbs and increase of all the deep reflexes occurs primarily and prevents the patient using the limbs, and after some months atrophy comes on in the tongue and hand muscles.

Cases of bulbar paralysis are very emotional, easily laughing or crying, otherwise their intelligence is good and they understand everything that is said to them.

Pathology.—In progressive muscular atrophy in the spinal cord the cells of the anterior horns have either atrophied and disappeared, or have lost their processes and are angular or globular, while the connective tissue in the grey matter is increased and the vessels are dilated. This is especially the case in the cervical enlargement of the cord, and if the legs are not wasted the cells of the lumbar cord are not affected. In the lateral columns there is no sclerosis in pure cases of progressive muscular atrophy.

In bulbar paralysis the cells of the spinal accessory, and hypoglossal nuclei, which probably also supply the lower face muscles through the facial nerve, and occasionally the cells of the seventh and fifth nuclei, have been found to be atrophied and absent.

In amyotrophic lateral sclerosis, besides the atrophy of the anterior cornual cells, the degeneration in the lateral columns is very marked, and has been traced up the pyramidal tract to the cortex cerebri, in which also degeneration of cells has been found (Marie).

In all three diseases the muscles waste, become pale, lose their striation, and undergo fatty degeneration with increase of the nuclei of the muscle sheath.

In these three diseases the lesion consists of a slow degeneration of the motor cells of the spinal cord and bulb, with the addition in the third case of sclerosis of the lateral columns and pyramidal tracts with sometimes degeneration of the cells of the motor cortex in amyotrophic lateral sclerosis. In the first two cases the lower motor segment is affected—from the anterior horn, or bulbar, cell to the muscle, while in the last all the motor tract from cortex to the muscles is involved, including the upper motor segment—cortex to end of lateral column fibre—as well as the lower segment.[1]

Prognosis is always unfavourable as these diseases tend to progress, but some cases remain stationary when they have reached a certain point; bulbar symptoms make the prognosis worse, owing to the liability to death from choking or from bronchitis and pneumonia due to deficient muscular power, so that these cases seldom live more than two years.

Treatment.—Besides general treatment, the main point in bulbar cases is the feeding; the food should be semi-solid rather than liquid, and the patient should be carefully fed with a spoon, or if this be impossible nasal feeding by a soft tube may be required. Very great attention is required in the clothing, so as to prevent catching cold.

With regard to internal remedies, phosphorus, arsenic, nux vomica, and cod-liver oil are indicated; but according to Gowers the best results are obtained by the hypodermic injection of strychnia nitrate gr. $\frac{1}{100}$ up to $\frac{1}{40}$ once a day into the affected muscles, which succeeds by subcutaneous injection, where given by the mouth it has failed to stop the course of the disease. In amyotrophic lateral sclerosis, strychnia would increase the spasm and is counter-indicated. In two cases of muscular atrophy I have seen, this treatment failed to arrest the disease.

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[1] In amyotrophic lateral sclerosis the first symptom will be atrophy or rigidity according as the lower or the upper motor segment is first affected (fig. 13, *a.h.c.*, *m.n.*, M., or *c.m.*, *p.t.*, *l.c.*).

It is doubtful whether electrical treatment has any certain influence, but still it may have some effect in keeping up the nutrition of the muscles; the constant current should be used, the positive pole being applied to the spine, and the negative being passed over the affected muscles, and using a current which is just able to cause contraction of the muscles. Massage and rubbing of the muscles may be tried for a time, in the hope of improving the nutrition.

Pseudo-hypertrophic Paralysis.

This disease and the one following are primary diseases of the muscles, and are hence called myopathies, and are really not diseases of the nervous system; but as they were formerly considered to be due to a spinal cord lesion, and as they resemble in many points true spinal muscular atrophies, they are still retained here. Pseudo-hypertrophic paralysis is characterised by symmetrical weakness of the muscles, which are either hypertrophied or atrophied, and this is associated with late contracture of the joints. It differs from spinal muscular atrophy in the different distribution of the affected muscles, and in the absence of fibrillar tremors and of the electrical reaction of degeneration.

Causes.—The disease is distinctly hereditary, and although it affects the males almost exclusively it is transmitted through the females. Thus all the boys of a family may have the disease and the sisters escape, but the latter, if they married, might transmit the disease to their sons. The usual *age* of onset is from four to ten years old.

Symptoms.—The child's parents first notice that he has some difficulty in walking and easily gets tired, also that he is always falling down, and that he gradually gets worse, but they do not notice any enlargement of the limbs.

The calf muscles are usually the first to be involved, and on examining them they are felt to be enlarged and harder and firmer than natural, and at the same time the walk becomes peculiar, the boy keeps his feet wide apart, and rolls from side to side as he brings the legs forward, and the knee is often projected in front of the foot. The peculiar rolling walk is due to the weakness of the glutei, so that the patient has to pitch the pelvis over the head of the femur as each step is taken; and also, owing to the weakness of the extensors of the knees and of the toes, the knee comes forwards in front of the foot, and then the leg is thrown forwards and the knee is straightened by planting the foot on the ground; going up and down stairs is also very difficult. In early cases, in spite of the large calf muscles, the patient cannot stand on tiptoe. In a typical case, in addition to the calves, the extensor cruris especially the vastus externus, and gluteus maximus are enlarged, while the anterior tibial muscles, the peronei and the flexors of the knee are often atrophied. In the upper limb, the deltoid, supra- and infra-spinati, and triceps are enlarged; while the sternal half of the pectoralis major, the latissimus dorsi, and the biceps are wasted, and the first two muscles sometimes completely disappear, so that the anterior axillary fold passes to the upper end of the sternum. In the back the erector spinæ is weak, and is sometimes hypertrophied.

There is no fibrillar twitching in the affected muscles, and their irritability to percussion is not increased. The knee jerk is lost as soon as the extensor cruris is involved. There is no anæsthesia, and the sphincters are normal.

The attitude of the trunk in standing is peculiar; owing to the paralysis of the glutei the pelvis is rotated very much forward, and to counteract this position the spine is bent concave backwards in the lumbar region, so that a plumb line hung from between the scapulæ falls well behind the sacrum, and if the patient once allows the upper part of his trunk to come forward so that his centre of gravity is in front of his base line he must fall down forwards. On sitting down this lordosis disappears, and is replaced by cyphosis, due to weakness of the erector spinæ. In getting up off the ground, when lying on his back, for the same reason, *i.e.* from weakness of the glutei and erector spinæ, he has first of all to roll on to his side, flexing his thighs under him, and then to get on to the hands and knees; he then has to extend the knees so as to be on the hands and feet, he suddenly transfers one hand to the knee, and thereby gets the spine straight with the other hand transferred to the knee, lastly he, so to say, climbs up his thighs with his hands, and thus manages to extend the hips until he attains the erect posture. Though this procedure is very frequently present in this disease, it is by no means diagnostic; it simply means that there is paralysis of the glutei or erector spinæ, or both.

The muscles, both those which are hypertrophied and those which are atrophied, become more feeble till they lose all power of contracting. The muscles of the forearm and of the hand are rarely affected.

Although in different cases the same muscle, as the deltoid, always hypertrophies, or as the latissimus dorsi always atrophies, there are some cases in which there is no initial hypertrophy but atrophy throughout, and which must be regarded as varieties of the same disease.

Later on in the disease the hypertrophied muscles, especially those of the calves, diminish in size, and their fibrous tissue becomes contracted, and produces deformities of the feet as talipes equinus.

With regard to electric reactions, the muscles respond to the faradic and constant current as long as there are any muscle fibres left, but though the current has to be increased there is no reaction of degeneration.

Pathology.—As no definite changes are found in the spinal cord or in the motor nerves, the disease is considered to be primarily in the muscles, which are pale and yellowish in colour, and are found to consist of masses of fat cells and fibrous tissue, with here and there a few muscle fibres, which may show granular or fatty degeneration. It seems probable that there is a congenital tendency to a primary interstitial fibrous overgrowth causing wasting of the muscle fibres in the muscles which atrophy; this overgrowth is associated with the deposit of fat in certain muscles which hypertrophy, and this produces the enlargement, while the absorption of the fat causes the subsequent diminution of these muscles. The hypertrophy is also due to absolute increase in the size of some muscle fibrils.

The distribution of the muscles affected and the grouping of those which atrophy and those which hypertrophy are quite different to that of spinal muscular atrophy.

Prognosis is very unfavourable, especially when the disease begins early; and there are, roughly speaking, three stages: the first stage when the child can get up off the floor unaided; the second when he can still walk, but cannot get up off the floor; and the last when he cannot walk or stand and gets contractures. When this condition is reached, it is rare for the case to live more than a few years, death occurring from bronchitis or pneumonia, owing to the weakened action of the respiratory muscles, before the child grows up.

Treatment is not able to arrest the progress of the disease; but it may perhaps be retarded by cod-liver oil, and tonics with phosphorus and arsenic. Great care should be taken against cold; but the child should be encouraged to get about as long as possible, as the disease progresses faster when the last stage is reached, and for this end gymnastic exercises with the limbs is very useful. Massage and electricity may retard the contractions, though they cannot be expected to cure the disease. Tenotomy of the tendo Achillis should be recommended, if there is sufficient power in the other muscles to use the leg when the position of the foot is corrected, otherwise it would be useless.

Simple Idiopathic Muscular Atrophy.

This, like the last, is a primary disease of the muscles, and is characterised by a gradual wasting of certain muscles with little or no hypertrophy, no fibrillar tremor, and no electrical reaction of degeneration. Several types have been described, but the most common is the one here given, and it was first described by Erb in 1884 as a "juvenile form" of muscular atrophy of the upper arm type.

Causes.—The family hereditary tendency is present in a certain number of cases and extends to former generations, but in some no such tendency can be found. In contrast to pseudo-hypertrophic paralysis, both sexes are affected about equally, and the age of onset is seldom before fifteen years, and may be as late as thirty or forty. No exciting cause can be definitely made out.

Symptoms are symmetrical, though one side may be affected first. The onset is gradual, with wasting and weakness of certain muscles ; and the first to be affected are the biceps, brachialis anticus, triceps, and the supinator longus, while the other muscles of the forearm and those of the hand as a rule escape. Of the other muscles, which are involved later, the pectoralis major, serratus magnus, latissimus dorsi, rhomboid, sterno-mastoids, and trapezius, wholly or partially, are wasted ; in the trapezius the superior fibres may be completely atrophied, a condition practically never seen in spinal muscular atrophy. The deltoid and supra- and infra-spinati usually escape. In the lower limbs the glutei, the flexors of the hip, and the extensors of the knee are wasted and weak, while the muscles below the knee are often not involved, or the dorsal-flexors of the ankle may be weak. The erectores spinæ are often much affected, and when the glutei are also much involved a very marked lordosis is produced ; if the recti abdominis are also weak, the lordosis persists when the patient sits down. The intercostals are sometimes affected.

There is considerable variety in the muscles affected, but there is never any fibrillar contractions and no electrical reaction of degeneration, though strong currents may be required to produce a contraction. The attitude, gait, and manner of getting off the ground may be the same as in pseudo-hypertrophic paralysis.

The knee jerk is never increased, but it may be lost if the extensor cruris is much wasted. There is no anæsthesia, and no affection of the sphincters.

In addition to the above symptoms, cases have been described by Landouzy and Dejerine in which the face is involved. The eyelids cannot be completely closed, and soap gets in on washing the face, but the muscles moving the eyeballs escape, the orbicularis palpebrarum being alone affected; there is also difficulty in elevating the angles of the mouth and in whistling, while the mouth is not closed properly, and the lips pout and are everted. This is due to the orbicularis oris and the levator labii superioris being weak. The tongue and the soft palate, the muscles of mastication and of swallowing and the vocal cords are not affected.

Pathology.—The spinal cord is normal, and the atrophy of the muscles is not attended by the overgrowth of connective tissue and the deposit of fat, seen in pseudo-hypertrophic paralysis. The size of the muscle fibres may be increased or diminished, they lose their striation, and show signs of fatty degeneration.

Prognosis.—This is very much more favourable than in pseudo-hypertrophic paralysis, and also than spinal muscular atrophy. The wasting progresses for a certain number of years (how long in any given case cannot be foretold), and then remains stationary; and though the wasted muscles do not recover, the patient does not get any worse—a case recently examined was in the same condition as when I saw him thirteen years ago. Cases do not die directly from the disease, but they may succumb to pulmonary diseases owing to deficient respiratory power. For diagnosis, see p. 190.

Treatment is the same as pseudo-hypertrophic paralysis, muscular exercises, massage, and the constant current, with tonics, being most useful; but as the disease tends to a spontaneous arrest, it is doubtful if treatment has much effect in producing this result.

In addition to the types given above, another form has been described by Charcot and Marie, and also by Tooth, which begins symmetrically in the peroneal muscles or extensor digitorum or extensor pollicis. It is sometimes hereditary, and occurs most in males about the age of puberty. After the peronei, the calf and then the thigh muscles are attacked, and in the upper limb the muscles of the hand are first affected. Some fibrillar twitchings are observed, and the muscles may show the electrical reaction of degeneration, and sometimes there is slight anæsthesia. The knee jerks are lost if the extensor cruris is involved. According to Tooth, these anomalous cases are probably due to a neuritis.

Myotonia Congenita (or Thomsen's Disease).

This name was given to a disease which was most accurately described by Thomsen, who suffered from it.

It is a peculiar spasmodic condition of the muscles, which comes on when the person, after resting, suddenly attempts to make a voluntary movement; the muscles become rigid and completely prevent the action for some minutes. It is often hereditary, and it is met with in both sexes, and in some cases the disease has been noted from childhood, but it is very rare in this country. As long as the muscles are kept moving the spasm does not come on. The muscles of the limbs are the most affected, and there is no wasting. The behaviour of the muscles to electricity is peculiar, and to a continued faradisation a prolonged tonic contraction of the muscle is produced, lasting several seconds, while the irritability of the muscles is increased to the constant current and to direct percussion.

Excision of pieces of muscles during life shows that the fibres are hypertrophied (Erb). The first post-mortem (1893) on a case showed the muscles to be very large and the fibres to be irregular in shape and size, while in places the fibres were atrophied with overgrowth of the connective tissue. There was no lesion in the peripheral nerves or central nervous system (Dejerine and Sottas). No treatment except exercise is recommended.

CHAPTER X.

TUMOURS OF THE SPINAL CORD.

THIS includes all tumours within the dura mater, whether in the substance of the cord or outside it.

Pathology.—Of tumours *outside the cord*, growing from the membranes or sheaths of the nerve roots, the most frequent are syphiloma, sarcoma, myxoma, fibroma, while tubercular and parasitic cases are less frequent. In the *substance of the cord*, glioma and syphiloma, myxoma, sarcoma, and tubercular growths, are most common. The growths may be single or multiple, and the latter occur especially as sarcomata or syphilomata growing on the nerves, and as tubercular within the cord. The size of a tumour outside the cord cannot well exceed half an inch in diameter, though it may be much longer; but within the cord a glioma round the central canal may extend the whole length of the cord. The cord is compressed and diminished in size by tumours outside it, and it is necessarily enlarged by growths within its substance. It is softened at the seat of compression by tumours external to it, and undergoes pressure myelitis, which is sometimes attended by secondary ascending and descending degenerations. With regard to the starting-point within the cord, gliomata often begin in the ependyma round the central canal, while syphilitic tumours grow in from the pia mater investing the cord ; these growths gradually invade the surrounding tissues, while sarcomata are either infiltrating or circumscribed and are then surrounded by a zone of softened tissue.

Symptoms are due to compression of the cord from without, or to absorption of the tissue by the infiltrating growth within the cord.

In the case of tumours growing outside the cord the symptoms are those of irritation of the spinal roots, followed by paralysis of the roots and of the part of the cord compressed by the growth.

The onset is gradual, and the chief symptom is a localised, very severe *pain*, which may be shooting, burning, cutting, and it occurs at a given level either round one half of the trunk or in one limb ; it is nearly always unilateral, and is much increased by movement, but to a less degree than in growths outside the membranes. Following on this pain there are other signs of root pressure, *i.e.* tingling, numbness, along or a little below the seat of the severe pain ; or there may be hyperæsthesia or a feeling of constriction and extreme tenderness in the skin, and muscular spasm and contracture of the muscles, supplied by the compressed roots. There is also tenderness to percussion and pressure over the vertebral spine at the level of the growth.

If the growth continue to enlarge, the above symptoms are followed by paralysis of the roots which were previously irritated, and of the part of the body supplied by the cord below the growth, *i.e.* one arm or one leg ; later on paralysis of the limb of the opposite side is caused by the pressure, so that paraplegia is produced. The onset of these symptoms is as a rule gradual and progressive, but sometimes, owing to myelitis being set up, the paralysis comes on more acutely.

Sensation is affected rather later than motion, and varies in its position according to the seat of the lesion. With a growth pressing on one side of the cord below the mid-dorsal region, sensation is affected on the same side as motion; but if in the cervical region, sensation is said to be affected on the opposite side (Gowers).

Wasting of the muscles and electrical changes in them occur accordingly as the anterior roots or the anterior cornua supplying them are pressed upon; in the same way the knee jerk is lost when the lumbar enlargement is compressed, and it is increased when there is pressure on the cord above this point; the sphincters are also paralysed in the former condition, so that the urine runs away as fast as it enters the bladder. The superficial skin reflexes, corresponding to the level of the growth, are lost.

The symptoms gradually progress, and they are varied by attacks of myelitis caused by the pressure of the growth.

With regard to localisation of the different parts of the cord, with a tumour in the cauda equina there is bilateral loss of power in the legs and wasting with electrical reaction of degeneration in all the muscles of the lower limbs with preservation of the flexors of the hip and the sartorius, loss of sensation and severe pains over the sacrum and perinæum, loss of the plantar cutaneous reflex action and paralysis of the sphincters.

In pressure on the lumbar enlargement the sharp shooting pains are in the lower limb of the same side, with paralysis and wasting of the muscles supplied by the roots compressed, loss of the knee jerk, if the third and fourth lumbar roots be involved, anæsthesia in parts of the leg of the same side, with complete paralysis of the sphincters ; followed later by paralysis of the other leg.

In the dorsal region there is very severe persistent pain localised to a definite area extending from the spine to the middle line in front, corresponding to the cutaneous distribution of the dorsal root of one side which is compressed, with sometimes anæsthesia of this area, and later on paralysis of the leg of the same side.

In the cervical region severe pain is felt in the arm of the same side with atrophy of the muscles, or anæsthesia of the skin area, supplied by the compressed roots; later on paralysis of the leg of the same side ensues with increased knee jerk.

Multiple growths outside the cord are often impossible to diagnose, as the symptoms are like those of diseases within the cord ; one case of mine simulated syringo-myelia.

The above description applies chiefly to growths from the membranes outside the cord. When the growth begins in the substance of the cord, owing to the absence of pressure on the roots the sharp localised pain, tenderness, and muscular spasm are not so marked, the cord symptoms begin sooner and are more often bilateral ; atrophy of the muscles is more extensive owing to the anterior horns being more readily involved over a wider vertical extent, and sensation may be lost up to the area supplied by the highest level of the cord affected.

Diagnosis.—See p. 189.

Prognosis is always grave, and if the growth be in the substance of the cord necessarily fatal, and even when the growth is syphilitic it does not always yield to medical treatment. The average duration of intra-dural tumours is given as from one year to about two and a half years; but if the growth is outside the cord and intra-dural, the prognosis is very much more favourable, as it is possible that it can be removed.

Treatment.—This is different, according as the growth is outside the cord or in its substance, but in all cases wherever the tumour is a course of anti-syphilitic treatment should be carried out for from six weeks to two months even if there is no history of syphilis. Since the brilliantly successful case of Gowers and Horsley,[1] there should be no hesitation in operating on a case in which a tumour can be diagnosed with any certainty to be outside the cord substance and where anti-syphilitic treatment has failed to relieve the symptoms. The difficulty consists in localising the level of the growth, and for this purpose the highest level on the trunk in which there is any change in sensation should be taken as the guide, remembering that the spinal nerves come off from the cord one or even two vertebræ higher than the foramen of exit. For instance, in the case quoted above, the line of complete anæsthesia on the trunk was the fifth dorsal nerve, with some doubtful diminution at the level of the fourth dorsal nerve; at the operation the lamina of the second dorsal vertebra had to be removed before the growth was seen pressing on the side of the cord, and involving the upper part of the fourth dorsal nerve where it leaves the cord.

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[1] *Roy. Med. Chir. Trans.*, 1888.

Growths within the substance of the cord should be treated with iodide of potassium and mercury, even though syphilis is denied. Pain must be relieved by morphia, chloral, and bromide, and other sedatives; and great attention should be paid to drawing off the urine and to the position of the patient, to avoid cystitis and bed sores.

Another method of ascertaining the highest level is that used by Horsley; it consists in giving a small hypodermic injection of pilocarpine gr. $\frac{1}{10}$; owing to the paralysis of the vaso-motor nerves from the pressure of the tumour, the sweating will not occur in the skin area supplied by these nerves and will be limited by a line running round the trunk, which corresponds to the highest level of the skin area supplied by the affected cord.

SYRINGO-MYELIA.

(Cavities in the Cord.)

This, which was formerly looked upon as a very rare disease, is now found to be much more common. It is characterised clinically by a progressive atrophy of the hand muscles, by loss of sensations to pain and temperature with preservation of tactile sensation, and by trophic changes as painless whitlows, and pathologically by cavities within the cord.

Pathology.—In the embryo it will be remembered that the central canal of the spinal cord is formed by the cells growing up as a ridge on either side of the primitive groove, and the two sides meeting enclose a cavity, and form all the substance of the cord, except the posterior median columns. A layer of cells cuts off the true central canal from the rest of the cavity, which lies posteriorly between the posterior external columns; this cavity is filled up by a layer of embryonal cells, which form the posterior median columns. Cavities in the cord are considered to be caused by a congenital defect, and are found under the following conditions: (1) the central canal is dilated with or without an overgrowth of the ependyma; (2) the cavity between the posterior columns persists; (3) the embryonal tissue may remain as such in the posterior columns, and may degenerate, producing cavities; or (4) this tissue may undergo an abnormal growth, and cause a central glioma, which degenerates and softens at its centre, producing a cavity. Around the dilated central canal the overgrowth of the ependyma may be slight or may be excessive and form a tumour around the central canal, which can usually be distinguished by its ependymal lining from the cavities produced by degeneration. We have therefore two changes, (1) a cavity which may be the central canal or one quite distinct from it, and (2) an overgrowth of the ependyma or of the embryonal cells in the posterior columns which may form an actual gliomatous tumour. These cavities contain fluid, which distends them and produces pressure on the neighbouring grey matter.

The cervical region of the cord is more often affected than the dorsal and lumbar; but in some cases the glioma has extended to the floor of the fourth ventricle, producing symptoms in the face and scalp.

Symptoms.—Although the conditions must to some extent have existed during life, it is rare for anything abnormal to occur before puberty, while sometimes no symptoms have occurred till after fifty years.

The symptoms begin gradually, with some loss of sensation in one hand, followed soon by that of the other. Though tactile anæsthesia is the exception, the loss to heat and cold and to painful impressions is very marked, and may involve one or both upper limbs, and extend up the head; in fact, one of the statements often volunteered by the patients is that they burn their hands without feeling any pain. In one of my cases there was loss to pain and temperature in both upper limbs and on the trunk above the level of the second dorsal spine, and the whole of the neck and head except the face. The legs and trunk are less frequently involved. The loss to pain, heat, and cold is not always of the same extent and degree, but one may be more affected than the others.

Later on muscular atrophy of the hand muscles is developed, first on one side and then on the other, with fibrillar twitching, which is often exactly like that of progressive muscular atrophy, and is associated with electrical changes giving the mixed form of the reaction of degeneration; and this is followed by atrophy of other muscles of the upper limbs.

The trophic changes are painless whitlows, pemphigus, eczema, glossy condition of the skin of fingers, a peculiar thickening and shortening of the phalanges, cracked nails, and in some cases dislocations of the joints and changes in the long bones. In one case I published with Mr. Lunn there was dislocation of both wrists and both shoulders.

The pupils are in some cases unequal, due to the dilator nerves of the sympathetic, which come from the first or second dorsal roots, being involved and paralysed, and so cause contraction, and non-dilatation to shade, of the pupil corresponding to the side which is the more affected. The legs are usually not atrophied, and are more often rigid with increased knee jerk and ankle clonus, owing to descending lateral sclerosis. Lateral curvature has been observed, and has been considered due to weakness of the spinal muscles. The sphincters usually escape.

Rarely nystagmus has been seen on making the patient look to the extreme right or left, and in a few cases hemiatrophy of the tongue. The symptom of atrophy of the hand muscles is due to the gradual pressure of the dilated cavities or of the gliomatous growth on the anterior horn cells. The loss to painful and temperature impressions, and not to tactile, is probably due to the paths for the two former being in the grey matter, which is always more affected than the white columns. For diagnosis, see p. 189.

Prognosis.—The disease as a rule runs an extremely slow course, but when atrophy has occurred no improvement can be expected to ensue. Some cases end acutely in a few months.

Treatment is the same as that for progressive muscular atrophy, and it cannot do more than improve the general condition. Care should be taken to prevent wounds and burns of the analgesic limbs, as troublesome ulceration may ensue.

Another form of the disease is that called Morvan's disease, or panaritium analgicum, where there is loss to all forms of sensation with painless whitlows, and pathologically peripheral neuritis is superadded to the cord changes of syringo-myelia.

INJURIES TO THE CORD.

Besides fractures of the spinal column, which have been discussed on p. 115, the cord is liable to be damaged by blows on the back from heavy weights falling on it, from direct falls on to the back or the feet, or from severe concussion, as in railway accidents; in cases which have terminated fatally, laceration or softening of the cord, hæmorrhage within or outside the membranes, or signs of chronic myelitis have been found.

Symptoms.—The element of fright or shock is so often associated with these accidents, that it is very important to separate the objective from the subjective symptoms.

The symptoms may come on immediately after receiving the injury or concussion, and in that case, if the whole thickness of the cord is damaged, there will be complete loss of motion and sensation with paralysis of the sphincters, and loss of the knee jerk followed by excessive action unless the lumbar region is involved; or the symptoms are less severe at first, but they are liable to increase from myelitis secondary to the original lesion—these cases are often fatal in a few weeks.

In other cases the symptoms do not come on till after a few days, when tingling and sensory symptoms occur, passing on to symptoms of subacute myelitis. Owing to the number of cases which show only subjective symptoms after accidents, it is often exceedingly difficult to say how much is due to organic changes in the cord; but each case must be taken on its own merits, and the subject is more fully discussed in the last chapter of the book, as well as the question of diagnosis from purely functional symptoms.

Prognosis in the first class of cases is unfavourable for life or recovery, and becomes worse as long as the symptoms are progressing; if the cord is lacerated completely across, and if death does not occur from bed sores or loss of respiratory power, there is practically no hope for recovery of power in the legs.

In the second class the prognosis is that of subacute myelitis.

Treatment is that of acute or subacute myelitis, and rest is one of the most important aids, unless there are any signs of fracture or dislocation, when an operation cannot be recommended too soon. For the pain, which is often a prominent symptom later, the bromides with chloral hydrate, cannabis Indica, and counter-irritation by the actual cautery or blisters, are useful.

CHAPTER XI.

DIAGNOSIS OF DISEASES OF THE SPINAL CORD.

Spinal cord diseases, in contrast with cerebral diseases, have a great tendency to affect the two sides of the body symmetrically, with the exception of acute poliomyelitis.

The most important point in making a diagnosis is to ascertain the mode of onset, as in two cases presenting the same symptoms the history of the mode of onset may distinguish between two diseases having a totally different course and ending.

The onset may be (1) sudden, in which the lesion is probably vascular or traumatic; (2) rapid, with an acute inflammatory condition; or (3) gradual, showing a degenerative process.

(1) Diseases of the cord which begin with a sudden onset, are hæmorrhage into the membranes, hæmorrhage into the cord, laceration of the cord, and hæmorrhagic myelitis. In the first two, pyrexia is absent; in hæmorrhage into the membranes there is sudden, acute, localised pain in the back, with signs of irritation of the spinal nerves, as pains radiating along the course of the nerves involved, followed by paralysis

of them; in hæmorrhage into the cord the pain is less severe, and local paralysis rapidly spreading occurs at once without preceding irritation symptoms; in hæmorrhagic myelitis the sudden onset has been preceded by pyrexia and malaise. Traumatic cases are often attended by fracture of the spine.

(2) Among diseases with a rapid acute onset are:—

Acute meningitis.
Acute myelitis.
Acute polio-myelitis.
Multiple neuritis (not a spinal disease, but most conveniently mentioned here).
Acute ascending paralysis.

All but the last begin with pyrexia, rigors, and other signs of inflammation, and take some hours or a day or two for development.

Acute Meningitis is characterised by signs of irritation, very severe spinal pain radiating along the roots, hyperæsthesia, retraction of the spine with spasm and rigidity of the muscles, followed later by signs of paralysis. It has also to be diagnosed from tetanus, in which there is often a history of injury, trismus is frequently present, and pyrexia is absent.

In Acute Myelitis the pain is much less marked, there is no spasm, and paralysis begins sooner than in meningitis; it usually affects the legs most, it is bilateral, and the sphincters are sooner involved.

In Acute Polio-myelitis there is not much pain, but rather more than in acute myelitis, the onset is more rapid than in acute meningitis and myelitis, and all four limbs may be paralysed in one night. Myelitis in children is almost always in this form, which further differs from the two above diseases in presenting no anæsthesia, no affection of sphincters or very rarely, and in the irregular distribution of the muscles and limbs involved.

From cerebral infantile hemiplegia (*q.v.*) it is separated by the electrical reaction of degeneration of the wasted muscles, the loss of the knee jerk, and by the flail-like condition of the limb, any contracture from the unopposed healthy muscles being passive, and not an active spasm. Acute polio-myelitis occasionally occurs in adults.

In MULTIPLE NEURITIS there have been premonitory pains and needles and pins in the limbs ; the onset is often less acute than meningitis or myelitis, and may take several days to develop ; the pains are not so severe as in meningitis, and not spinal, but rather a tenderness of the peripheral nerves and muscles ; the paralysis differs from that of myelitis in picking out first the extensors of the wrists and feet instead of the whole of the lower limbs ; the knee jerk is lost early, and the muscles waste and give the electrical reactions of degeneration, which is not the case in myelitis, unless the brachial or lumbar enlargement is involved. The sphincters are also rarely affected. The alcoholic history also assists the diagnosis.

LANDRY'S PARALYSIS differs from all the foregoing in there being little or no pyrexia, no anæsthesia, no sphincter troubles, no electrical changes or wasting of muscles, but a paralysis which begins in the feet and ascends in a few hours till death is caused by failure ot respiration.

Spinal cord diseases have to be diagnosed from Hysterical Paralysis by the absence in the latter of incontinence of urine and of ankle clonus, and by normal electrical reactions (see "Hysteria").

(3) Diseases of the cord which begin with a gradual onset are rather more difficult to group; but the following may be taken together, (*a*):—

Chronic myelitis.
Primary lateral sclerosis.
Compression of the cord from caries, etc.
Pachymeningitis hypertrophica.
Tumour of the spine.
Tumour of the cord.

The first two, in which pain is not a leading symptom but rigidity occurs, may be considered together.

Chronic Myelitis, if it follows the acute form, is readily diagnosed, but it may come on with gradual weakness and loss of sensation in the legs with some girdle pain, and, later, affection of the sphincters with rigidity of limbs; while in Primary Lateral Sclerosis the stiffness is more marked as a primary symptom, and the deep reflexes are more active all over the body, there is no anæsthesia and no girdle pain, and the sphincters are often not affected.

The other members of this group are characterised by a gradual onset, with severe localised pain, which radiates round one or both sides of the trunk. The severity of the pain and its increase by movement of the spine assist the diagnosis. Compression due to spinal caries, in the absence of the diagnostic symptom of projection of the vertebral spines, is characterised by the pain being less severe and less affected by movement than the other members of the group, and by the paralysis being as a rule bilateral from the first. In HYPERTROPHIC PACHYMENINGITIS the pains are bilateral, very severe, increased by movement, radiate down the limbs involving several roots, especially the lower brachial, and the pains are followed by paralysis of motion and sensation with wasting of muscles.

TUMOURS OF THE SPINE and of the CORD are distinguished from the last disease by a very sharp, persistent pain in one place, usually unilateral, radiating along one root and increased by movement, with local rigidity or spasm of the muscles supplied by the affected root. This is followed by progressive paralysis of the limb on the same side as the pain, and after an interval of some weeks of the other limb, and later with rigidity and increased knee jerks and gradual affection of the sphincters. With tumour of the spine there is rather more pain on movement than with a growth inside the spinal membranes, owing to the roots being more irritated.

(*b*) The following group is characterised by a gradual onset, with weakness and for the most part wasting of the muscles, but with little or no pain :—

Syringo-myelia.
Progressive spinal muscular atrophy.
Amyotrophic lateral sclerosis.
Idiopathic muscular atrophy. } Not spinal cord
Pseudo-hypertrophic paralysis. } diseases.

SYRINGO-MYELIA is put first as being frequently caused by a neoplasm of the cord. It is therefore allied to the last group. Syringo-myelia and Progressive Spinal Atrophy are characterised by gradual wasting and weakness of the muscles, beginning in the small hand muscles with fibrillar twitchings and loss of electrical reactions; but while in progressive muscular atrophy there is no affection of sensation, in syringo-myelia sensation for pain and temperature is much affected or completely lost, and this is associated with normal or very slightly affected tactile sensation, and at the same time marked trophic changes, as whitlows and dislocations, occur.

Amyotrophic Lateral Sclerosis is progressive muscular atrophy with rigidity of the lower limbs and increase of the "tendon reflexes" everywhere.

Idiopathic Muscular Atrophy in adults and Pseudo-hypertrophic Paralysis in children have a gradual onset, with atrophy of some, and hypertrophy and hardening of other muscles, and they differ from the two foregoing diseases in beginning in the trunk and upper arm and thigh muscles rather than in the hand, in having a family history, and in having no fibrillar twitchings and no electrical degenerative reactions. In the absence of hypertrophy the diagnosis of pseudo-hypertrophic paralysis is made probable by the peculiar walk, the wasting of the sternal part of the pectoralis major, and the absence of the knee jerk. The grouping of the affected muscles in these two diseases is quite different to that of true spinal muscular atrophy.

(*c*) In the last group, which is characterised by symptoms of ataxia and inco-ordination rather than by those of wasting and motor paralysis, are :—

Locomotor ataxia.
Ataxic paraplegia.
Hereditary ataxia.

In Locomotor Ataxia the chief diagnostic symptoms are the characteristic sharp pains, the lost knee jerks, the non-reaction of the pupils to light, and the inco-ordination when the eyes are closed, with affection of the sphincters. In Ataxic Paraplegia there is inco-ordination followed by a spastic condition, the pains are slight and dull, the knee jerks are excessive with ankle clonus, the pupils react to light, but the ataxia is well marked; while in Hereditary Ataxia the disease begins in children or at puberty, at an age earlier than in the two former diseases, and attacks several members of one family. There are usually no pains, the knee jerks are absent, the pupils react to light; but the most marked symptom is the inco-ordination with the closed eyes, and this, taken with nystagmus, a blurred speech, occasionally lateral curvature and talipes equinus, separates the disease from the two foregoing and from disseminated sclerosis. Also in the last-named disease the knee jerk is excessive, and the inco-ordination is not increased by closing the eyes.

Locomotor ataxia has also to be diagnosed (*a*) from multiple neuritis, which it resembles very much, but in the former disease there is not the complete symmetrical paralysis with the electrical changes and extreme tenderness and wasting of the muscles, and in the latter the pains are not so sudden, the pupils react to light, and the sphincters are very rarely affected; (*b*) from general paralysis of the insane by the absence of facial tremor, of overaction on voluntary effort, and of a slurred speech, but as the two diseases may coexist the differential diagnosis is often impossible; (*c*) from tumor cerebelli in those cases where there is optic atrophy and loss of the pupil light reaction, but in cerebellar cases the staggering is more reeling and is not increased by closing the eyes, the pains and anæsthesia of the limbs are absent, there is more headache and vomiting, and the knee jerk is also increased, but occasionally it may be absent; (*d*) from diphtheritic paralysis, where the absence of lightning pains and the distinct loss of power in the limbs with frequent paralysis of the soft palate are important symptoms.

Ataxic paraplegia has further to be separated from primary lateral sclerosis, in which there is no incoordination, from cerebellar tumour and from chronic myelitis.

CHAPTER XII.

DISEASES OF THE CRANIAL NERVES.

THE cranial nerves are so liable to be affected independently of other diseases, and also as the results of diseases, as meningitis and tumours, that it is necessary to describe them separately.

OLFACTORY OR FIRST CRANIAL NERVE.

Loss of smell for odours as assafœtida, oil of cloves, may be due to disease of the mucous membrane or to trophic changes due to affection of the fifth nerve, but loss of smell is also due to injury to the olfactory bulbs from fractures of the skull, meningitis, or pressure from intra-cranial tumours or hydrocephalus. In the case of tumours, loss of smell of one side would be in favour of one frontal lobe being involved, but bilateral loss of smell may be caused by intra-cranial tumours anywhere, and it has no localising value. Subjective olfactory sensations have been observed before epileptic attacks caused by a tumour in the anterior tip of the temporo-sphenoidal convolution, which is considered to be the olfactory centre, as in a case observed by Dr. Hughlings Jackson and myself.

OPTIC OR SECOND CRANIAL NERVE.

The path of the optical fibres (see fig. 10) has been fully described on p. 27.

Lesions in the optical fibres may occur—(1) In the optic nerve itself, from tumours at the back of the orbit, from aneurysms, abscess or fracture of the skull; the symptoms will be a gradual loss of sight of that eye, with peripheral limitation of the field of vision, with optic atrophy and loss of the pupil reflex to direct stimulus of light, but with retention of reflex consensual contraction to light acting on the pupil of the sound eye.

(2) At the chiasma, especially from growths in the pituitary body, which may be simple, syphilitic, or tubercular, or from the pressure of a hydrocephalus. The symptoms differ according to whether the central part or the lateral parts of the chiasma are involved. The former, which is the more frequent, produces temporal hemianopia, *i.e.* loss of vision in the right field of the right eye and in the left field of the left eye, with retention of the fields on the nasal side of the middle line; the latter, which is very rare, and of which I have published (with Mr. Lang) an example occurring in a case of locomotor ataxia with optic atrophy (fig. 19), in which probably the lateral fibres were most affected, produces nasal hemianopia in both eyes. The same condition might be produced by two tumours, one pressing on either side of the chiasma. Optic atrophy ensues in most cases, and both eyes may become completely blind in time.

(3) From the chiasma to the occipital region, in-

cluding the optic tract, the most posterior part of the internal capsule, the optic radiations, and the outer and median surfaces of the occipital lobe; the symptom observed is what is termed homonymous hemianopia (fig. 18)—*i.e.* with lesion of any of the above parts on the right side vision is lost for all objects on the left (opposite) side of the middle line of both eyes. Though the vertical dividing line corresponds to the point of fixation of the eye, this point itself is usually spared, as shown in the diagram (fig. 18), vision extending 5° to 10° on the blind side of this point; this has been considered due to the macula lutea of the retina of each eye sending fibres to each optic tract, and so on to each occipital lobe, but the explanation is not satisfactory. Lesions of the optic tract are produced by tumours in the middle fossa of the base of the skull, or by meningitis especially syphilitic, while the rest of the visual path, including the occipital cortex, may be the seat of hæmorrhage, softening, abscess, or tumours. As the optic tract contains fibres going from the retina to the corpora quadrigemina, which is considered to be the reflex centre for the contraction of the pupils to light, a lesion of the right tract by stopping the stimulus from the right half of each retina will prevent the pupils reacting to a light which comes to them sideways from the left (*i.e.* blind) half of the field of vision, whereas they will react to light coming from the right fields, but the observation is of no value unless there is a distinct difference—this condition is called the hemiopic pupil; no such difference in the action of the pupils is obtained in lesions of the internal capsule, optic radiations, or occipital lobe. Hemianopia due to a lesion of the internal capsule is especially associated with hemianæsthesia. In rare cases of lesion of part of the occipital lobe (cuneus) one-quarter of each field is lost in place of one-half.

(4) Lesions of the supra-marginal and angular gyri are considered by many to produce what is known as "crossed amblyopia"—*i.e.* with a lesion of the left side, a concentric diminution of the field of the right eye to 5° or 10°, and of the left eye to 20° or 30°; but if the lesion be deeper and involve the subjacent optic radiations, right hemianopia would be caused. These cases are associated with word blindness (see "Aphasia"), and are produced by softening, tumours, or abscess.

Crossed amblyopia also occurs in hysteria, and is associated with complete loss of colour sense (achromatopsia) in the most affected eye.

Optic neuritis.—This may affect one eye or both, and in the former case it is most frequently caused by tumours pressing on the optic nerve at the back of the orbit, and is associated with rapid loss of sight of that eye and loss of the pupil reflex to light. Double optic neuritis is produced by intra-cranial tumours, and by acute meningitis; it also occurs in albuminuria, in lead poisoning, and in extreme anæmia; one eye is often affected before the other, and marked neuritis may be present without vision being impaired.

Optic atrophy.—This occurs as a primary condition in locomotor ataxia, disseminated sclerosis, and in general paralysis as grey atrophy, and secondary to optic neuritis as white atrophy

OCULAR NERVES (THIRD, FOURTH, AND SIXTH).

The ocular muscles which are found to be paralysed differ in their arrangement according to whether the lesion is in (1) the several nerves in their course from the medulla, where they arise, through the cranial cavity and orbit to the muscles; (2) the nuclei of these nerves in the pons and medulla; (3) the cortex cerebri itself, or the motor fibres passing from this through the internal capsule to the nuclei.

(1) In the first case we have paralysis of the muscles according to their anatomical distribution similar to the paralysis of, for example, the ulnar nerve in the arm. When the whole third nerve is affected, there is ptosis or drooping of the eyelid, and paralysis of all the external muscles of the eyeball, except the superior oblique (fourth nerve) and the external rectus (sixth), dilatation of the pupil with loss of contraction to light and loss of accommodation; the only movement possible is slightly downwards with rotation (see p. 59), and outwards, and the eye is permanently drawn outwards. Paralysis of the fourth nerve affecting the superior oblique muscle is best detected by the wheel-like rotation of the eyeball in looking down, produced by the inferior rectus, which rotates outwards the upper edge of the iris, as well as by the diplopia and position of the false image (p. 63). Paralysis of the sixth, with loss of power in the external rectus, causes inability to turn the eye outwards—say to the right—past the middle line, and there is diplopia on looking to the right side of the middle line, the images on the hori-

zontal plane being simple, erect, and diverging the more the object is moved to the right side.

The ocular nerves may be the seat of neuritis, which may be rheumatic from cold, syphilitic, or rarely diphtheritic, or they may be compressed by growths in the orbit or in the pituitary fossa, by aneurysms of the carotid or thrombosis of the cavernous sinus,—the third and fourth course in the outer wall of the sinus, and the sixth comes across its cavity—or by tumours and acute meningitis in the cranial cavity, or be injured by extravasation of blood after blows on the head or fractures of the skull. Of all single muscles the external rectus is most commonly affected, and one or both sixth nerves may be paralysed from increased intra-cranial pressure without being directly involved.

A lesion such as hæmorrhage or softening might affect the third and sixth nerves within the medulla between their superficial origin and their nuclei; the paralysis of the ocular muscles is then associated with loss of power in the opposite arm and leg, due to the pyramidal fibres being affected above their decussation.

(2) The third nerve passes through the crus cerebri at the superior border of the pons to its nucleus along the floor of the aqueduct of Sylvius, and just below it is the fourth nucleus, the nerve from which leaves the pons at its posterior aspect, decussates with its fellow, and winds round outside the crus cerebri to the outer wall of the cavernous sinus. The sixth nerve emerges at the lower border of the pons from its nucleus in the floor of the upper part of the fourth ventricle (just above the level of its widest diameter). The cells of the third nucleus for the different muscles

are probably arranged in order from above down (Kahler and Pick), so that the cells presiding over accommodation and the action of the pupil are the most superior, farther down are those for the levator palpebræ and the internal rectus, and farther back the rectus superior and obliquus inferior, while the rectus inferior is the lowest and is next to the fourth nucleus which supplies the superior oblique.

Internal ocular muscles.—Paralysis of the ciliary muscle with loss of accommodation may occur from a lesion affecting the whole third nerve or the anterior part of its nucleus. Paralysis of the contraction fibres of the iris occurs when the third nerve is affected, and the power of dilatation is lost when the sympathetic fibres (which come off from the second dorsal spinal nerve) are involved. Contraction of the pupil occurs when the eye is accommodated, and contraction and accommodation may be lost together or independently.

Loss of reaction to light is caused by disease of the anterior part of the third nucleus, and is especially common in locomotor ataxia and general paralysis without loss of the reaction to accommodation. Normally the reaction is bilateral and consensual—*i.e.* light thrown on to one retina acts through one optic nerve, through both (?) optic tracts, the corpora quadrigemina, the anterior part of the third nucleus of either side, and so through the third nerves and ciliary nerves to the iris of both eyes.

A lesion of the sixth nucleus (as a tumour or rarely a hæmorrhage) differs from one of the sixth nerve in that, in addition to the external rectus of the same side being paralysed, the internal rectus of the opposite side is also paralysed, so that both eyes are conjugately deviated to the opposite side *away* from the side of the lesion, being pulled over by the unaffected muscles. This is explained by the sixth nucleus being joined to the part of the opposite third nucleus which supplies the opposite internal rectus, and also by the fibres coming from the motor cortex going first to the sixth nucleus (see fig. 4).

Owing to the fibres of the facial (seventh) nerve winding round the sixth nucleus, the whole facial nerve is often paralysed on the same side when the sixth nucleus is the seat of lesion ; while if the lesion is sufficiently large, from the pyramidal fibres being involved above their decussation, the opposite arm and leg are paralysed. In these cases the power of convergence of the affected internal rectus with the opposite internal rectus is possible, but not that of horizontal movement conjugate with the external rectus of the other eye.

The most common form of nuclear paralysis is what is known as ophthalmoplegia externa, due to a gradual degeneration of the nuclei of the third, fourth, and sixth cranial nerves. The muscles are usually affected in the order of their association (*i.e.* the levator palpebræ and superior rectus together), and often symmetrically in the two eyes, but sometimes irregularly ; finally in advanced cases there is double ptosis, usually not complete, and complete fixation of both eyeballs, of which condition, owing to the very gradual onset, the patient may be quite unaware, but which may be recognised by the continual necessity of moving the head in looking in different directions.

In some cases the pupils may not react to light or accommodation (ophthalmoplegia interna); the orbicularis palpebrarum is also very weak in these cases, so that the patient cannot keep the eyelids from being forcibly opened. Ophthalmoplegia is often associated with syphilis, locomotor ataxia, and general paralysis of the insane. Sudden and acute nuclear paralysis, due to vascular and inflammatory changes, have been described, but they are rare.

(3) The associated muscles of both eyes are paralysed from a lesion of the cortex or internal capsule, but never individual muscles. The most common form is that of the muscles which move the eyeballs conjugately to one side, and it is explained as follows. Starting from the centre for turning both eyes to the opposite side (p. 13, fig. 4) in the right cortex cerebri, the fibres pass down through the right internal capsule, through the crus cerebri, and cross over (where is not known for certain) to the left sixth nucleus; from there fibres go up (probably along the posterior longitudinal bundle), and cross over to that part of the right third nucleus which sends fibres to the right internal rectus. Hence a lesion of the left sixth nucleus, as shown above, produces conjugate deviation of both eyes to the right, and the same effect is caused by a lesion of the right cortex or internal capsule.

This is practically the only symptom of ocular paralysis obtained from a lesion of the cortex cerebri or of the motor tract, extending from the cortex down to where the fibres cross to the opposite fibres, and it is to be particularly noted that whereas in lesion of the sixth nucleus the eyes turn conjugately *away* from the lesion, in the case of the cortex and internal capsule the eyes turn *towards* the lesion. Theoretically the conjugate movements upwards and downwards of the eyes might be paralysed, but this does not occur except when both internal capsules are affected.

Prognosis of ocular paralyses depends very much whether they are due to lesions of the nerves or the nuclei; the prognosis for recovery is better for nerve lesions and for acute nuclear lesions, and worse for chronic progressive nuclear paralyses, from which it is not possible to expect improvement, but cases of ophthalmoplegia do become stationary and continue so for some years.

Treatment for acute nerve lesions consists in hot fomentations to the orbit and temporal region, followed by a blister over the temple, and in cases where syphilis is suspected mercury and iodide, while for the chronic form tonics as arsenic, strychnia, and iron. The constant current has been recommended, applied to the closed eyelids with a soft brush or the operator's finger, to which is attached the wire from the battery; but its advantage is very doubtful.

THE TRIGEMINAL OR FIFTH CRANIAL NERVE.

This nerve supplies the sensory fibres to the face according to the diagram at the end of the book. The second part also supplies the upper gums, the upper part of the inside of the cheek, the roof of the mouth, soft palate (?), tonsils, and upper part of pharynx; while the third part supplies the lower gums, tongue, floor of mouth, lower part of inside of cheek, salivary glands, and external auditory meatus, also the fibres for taste on the anterior edge and tip of the tongue. The motor part supplies the tensor tympani, the masseters, pterygoids, temporals, buccinator, mylo-hyoid, and anterior belly of the digastric. The nerve arises from the lateral surface of the pons, as a large sensory and a small motor branch ; traced through the pons to the posterior surface, the sensory root goes to the middle nucleus near the outer part of the floor of the fourth ventricle and to two roots, a superior and inferior, which extend in the pons and medulla through their whole length, while the motor fibres come from a nucleus placed internal to the middle nucleus.

The nerve may be affected (1) between the face, and its exit from the cranial cavity by neuritis, but this is rare, or by tumours ; (2) within the cranial cavity by tumours and aneurysms, at the base of the brain by meningitis, especially syphilitic, and by caries of the petrous part of the temporal bone, in the hollow of which the Gasserian ganglion lies ; (3) within the pons by local hæmorrhage, by softening or tumours affecting the nuclei or part of them, or the whole nerve as it passes through the pons, may be involved.

Symptoms, when the whole nerve is affected, are first severe burning pain, followed by anæsthesia over the whole part supplied by the nerve up to the middle line, with loss of the conjunctival, nasal, and palatal reflexes on that side, with loss of smell owing to dryness of the mucous membrane, later food collects between the cheek and the jaw, and the half of the tongue is furred, owing to insensibility of the cheek and the weakness of the masticating muscles.

Taste is lost everywhere on one side when the root of one fifth nerve is involved just outside the pons, but probably not when within the pons; it is lost on the tip and anterior edge when the chorda tympani—which, when traced centrally, goes from the gustatory branch of the fifth to the facial nerve in the middle ear, and thence the fibres pass by the Vidian nerve to the spheno-palatine ganglion of the second division of the fifth—is involved in middle-ear disease; it is lost on the posterior part of the tongue and palate when the third division of the fifth nerve is affected, the fibres reaching it from the tongue probably through the glosso-pharyngeal nerve, Jacobson's nerve, and the otic ganglion.

Trophic changes are produced by irritation of the Gasserian ganglion, causing cloudiness of the cornea leading to perforation, and later to complete disorganisation of the eyeball, while the secretions of the lachrymal and salivary glands are altered. Another condition, known as hemiatrophy of the face, causes gradual wasting of the skin and fat, and in some cases of the bones of the face of one side, without affecting the muscles or the cutaneous sensation; this is due to degeneration and atrophy of the superior root of the fifth nerve within the pons (Mendel). Herpes over the regions supplied by the branches of this nerve is due to irritation of the ganglion or some part of the nucleus in the medulla.

FACIAL OR SEVENTH CRANIAL NERVE.

Besides being the motor nerve of the face, it supplies also the stapedius, the stylo-hyoid, digastric (posterior belly), platysma, both parts of the occipito-frontalis muscles, and in part the buccinator. The soft palate is not supplied by the facial, but by the accessory to the vagus (*q.v.*).

The symptoms differ according to the position of the lesion, and it will be more convenient to examine the different positions from the periphery to the central parts.

The facial nerve may be affected (1) between the muscles and its exit from the stylo-mastoid foramen; (2) in the aqueduct of Fallopius of the temporal bone and as it passes through the middle ear between the cochlea and the vestibule; (3) in the internal auditory meatus, and from this to the medulla; (4) in the passage of the nerve through the medulla, where it has a winding course, at first backwards round the sixth nucleus and then forwards to the seventh nucleus in the anterior part of the medulla; (5) indirectly by a lesion between the nucleus and the opposite motor cortex in the opposite crus cerebri and internal capsule.

In the face (1) the nerve may be affected by cold, injuries, and by tumours in the parotid; (2) in the temporal bone by cold, sitting in a draught or in a railway carriage with the window open, by diseases of the middle ear, especially suppurating otitis media, fractures of the skull; (3) within the skull by meningitis especially syphilitic, or tumours; (4) inside the medulla by hæmorrhage, softening, or tumours; (5) and from a lesion in the crus cerebri, internal capsule, and cortex it forms part of the paralysis of hemiplegia.

Symptoms.—In all the regions above noted, except the last, there is complete paralysis of the whole of one side of the face; so that the brow of that side cannot be wrinkled, the eye is open and cannot be closed, the corner of the mouth cannot be elevated, retracted, or advanced to whistle, the raphe of the upper lip is drawn to the non-paralysed side, while food collects between the buccinator and the gums, the eye waters, and saliva runs out of the corner of the mouth. The paralysis is complete for voluntary as well as for emotional movements, as in laughing. The muscles lose their electrical reaction to faradisation, and give the reaction of degeneration to the galvanic current (p. 72). This forms what is known as Bell's paralysis. The symptoms present some points of difference according to the seat of the lesion.

In a lesion of the nerve in the face all the muscles may not be paralysed, if only certain branches of the nerve are involved. In the aqueduct of Fallopius, the commonest seat of "rheumatic" paralysis, there is in addition loss of taste on the anterior part of half the tongue—due to the chorda tympani being involved, which here leaves the facial nerve, crossing the tympanic cavity down to the Glaserian fissure on its way to join the gustatory branch of the fifth nerve (p. 204)—and sometimes along the posterior half as well. In the internal auditory meatus and within the skull taste may not be lost, but hearing is lost without any evidence of middle-ear disease. Within the medulla taste is not affected, but more commonly there is paralysis of the sixth nucleus (conjugate deviation of the eyes away from the lesion) and paralysis of the opposite arm and leg, giving rise to "crossed hemiplegia."

Above the seventh nucleus, paralysis of the face is of the "cerebral type," and is often part of a hemiplegia, and may be caused by a lesion of the opposite motor cortex, by hæmorrhage, softening, abscess, or tumour of the opposite internal capsule or crus cerebri—or, in other words, anywhere from the cortex down to the point where by a lesion the fibres cross over in the pons to reach the seventh nucleus. Here the paralysis differs from that in the nuclear and nerve lesions, in that the frontalis muscle acts equally well on the two sides, the opposite eyelids can be closed, though sometimes less powerfully than normal, the mouth can be pursed up to whistle, and though retraction and elevation of the corner of the mouth cannot be produced voluntarily, they occur at once when the patient laughs, the conjunctival reflex is not lost, and all the muscles react normally to both electric currents.

Facial paralysis due to nuclear or nerve lesion runs a different course according to its severity. In the rheumatic form the duration lasts from a week to several months. The prognosis is determined by the electric reactions. If the muscles never lose their faradic reaction, recovery will be certain, and the paralysis will last only a few weeks ; if the faradic reaction is lost, recovery is doubtful, but it is not hopeless as long as the muscles react to the constant current, but when this is lost there is no chance of recovery. In middle-ear disease and in lesions of the seventh nucleus, the paralysis is usually permanent, while recovery may occur from involvement of the nerve just outside the medulla, which may be syphilitic. In muscles which have been for some months paralysed a contracture takes place which diminishes the deformity, unless the contracture goes too far, when an excessive naso-labial fold may be produced on the paralysed side.

Treatment.—In the commonest form, the rheumatic, the patient should be kept indoors, and should take liq. am. acetatis, acetate of potash, and spiritus ætheris nitrosi, and later salicylate of soda in full doses ; the face, front of the ear, and the external meatus should be douched with water as hot as the patient can bear or be steamed over a jug of boiling water several times a day for the first two or three days, and then a blister is to be applied over the lower part of the mastoid process.

Electric treatment should not be employed till after ten days or a fortnight, when all acute symptoms will have subsided. The constant current should be used, the positive pole being placed behind the ear just below the mastoid process, and the muscles gently stroked with the negative, using a current just strong enough to produce a contraction without causing flashes of light in the eyes. It may be continued as long as the reaction to the constant current is retained. After a month tonics as quinine and strychnine may be given. In the relief of secondary contracture massage of the face may do good. If syphilis be suspected, and the nerve be involved in the cranial cavity, iodide of potassium in full doses is indicated.

FACIAL SPASM.

This occurs spontaneously, or as a reflex condition of neuralgia of the fifth nerve, and is usually a disease of middle life. It commonly affects the muscles of one side, the eyelids being tightly closed and the angle of the mouth elevated ; but in other cases the spasm may be bilateral, affecting the eyelids of both sides whenever the patient fixes the eyes. This occurred in a man who over-used the eyes in hammering copper articles. It seems probable that the spasm may occur from pressure on the facial nerve, reflexly from neuralgia or carious teeth, or centrally from a functional (as in the case referred to) or organic cause, as softening of the ascending frontal convolution (Berkeley, quoted by Gowers). The contractions occur momentarily every few seconds, but a spasm may be prolonged for several seconds, and is often attended by pain. The seat of the disease can only be conjectured from the muscles involved. If the whole of one side of the face, including the one-half of the frontalis, contracts, the lesion would be in the nerve or its nucleus; but when the frontalis or orbicularis palpebrarum of both sides contracts, it is probably in the cortex cerebri. As a rule, intermittent spasm means irregular action of grey matter, and not pressure on motor fibres.

Treatment.—In reflex conditions pressure on the branches of the fifth nerve in the face may stop the movement for the time, and in those cases carious teeth must be looked for and treated, or counter-irritation as blisters may be applied over the branches of the fifth nerve. Sedatives, as opium, morphia, or conium, will stop the spasm; but the use of the first two is of very doubtful advantage, as it must be prolonged.

The constant current has been tried with the positive pole over the muscles and the negative at the back of the neck, using a current, increased without breaks, as strong as the patient can bear.

THE AUDITORY OR EIGHTH CRANIAL NERVE.

This nerve has two functions: the fibres which come from the cochlea are for the purpose of hearing, while those which come from the semicircular canals are probably for informing the individual as to the position of his head in space. The functions of the auditory part of the nerve may be affected by loss of hearing, by increased power of hearing, and by irritation of the nerve causing subjective sensations as noises, tinnitus aurium, while lesions of the semicircular canals or the fibres coming from them produce vertigo with actual loss of balance.

The auditory apparatus may be affected in its different parts between the membrana tympani and the cortex cerebri, *viz.*—by disease of the membrana tympani, as perforation and destruction; by disease of the middle ear, as suppuration; by disease of the internal ear, which is usually secondary to middle-ear disease or due to syphilis; by lesions of the auditory nerve, as in syphilis, meningitis, or locomotor ataxia, as far as its nucleus, which is in the medulla near its junction with the pons; by lesions of the auditory fibres, which cross to the opposite side, and probably ascend in the tegmentum of the crus cerebri to the corpus geniculatum internum, and thence by the posterior part of the internal capsule to the auditory centre in the opposite first temporal convolution (see p. 25).

In deafness due to disease of the middle ear, sounds are not heard through the air (aerial conduction), but are heard through the bone when a sounding tuning-fork is placed on the mastoid process (perosseal conduction); normally a tuning-fork placed on the mastoid, when it ceases to be heard through the bones, can still be perceived through the air when held opposite the external meatus without touching (Rinné's test); failure to hear it shows disease of the middle ear or of the tympanum, or blocking of the external meatus by cerumen.

Disease of the internal ear and of the rest of the auditory apparatus between it and the cortex is shown by loss of perosseal conduction of hearing, and the localisation in the different parts of the auditory tract can only be surmised by other symptoms. Disease of the internal ear would probably be associated with tinnitus or vertigo; of the auditory nerve, with paralysis of the facial nerve (but this association is of diagnostic value only if middle-ear disease be excluded) and tinnitus; of the auditory nucleus, with paralysis of the opposite limbs; of the internal capsule, with hemianæsthesia and hemianopia on the same side as the loss of hearing; and of the cortex (on the left side) with word deafness besides loss of hearing in the right ear (see "Aphasia").

TINNITUS AURIUM.

This is a subjective symptom of irritation of the auditory apparatus, and besides being caused by diseases of the external and middle ear, it is also produced by disease of the internal ear, as in auditory vertigo (*q.v.*), by the state of the blood in anæmia, by intracranial aneurysm, by any exhausting disease, and by certain drugs as quinine, salicine. The character of the noises varies from hissing and roaring to musical sounds as bells; in some cases the noises are stopped by compression of the vertebral or carotid arteries, in other cases not; they may be intermittent or continuous. Hearing may be gradually lost or not affected.

As tinnitus is only a symptom, the cause must be ascertained. When due to anæmia or to aneurysm, the sound is intermittent, and there may be no deafness, while a continuous sound with deafness is more likely to be in the internal ear itself.

Treatment consists in counteracting the cause, such as anæmia, syphilis, gout, and by giving bromide of potassium, and using the constant current as in auditory vertigo. In all cases the ear should be examined for cerumen, etc., pressing on the tympanic membrane, and inflation of the tympanic cavity may be required.

THE GLOSSO-PHARYNGEAL OR NINTH NERVE.

This nerve is so seldom paralysed alone that it is difficult to know its exact functions; but lesions of it probably cause difficulty of swallowing, owing to paralysis of the stylo-pharyngeus, some loss of sensation of the upper part of the pharynx, and loss of taste in the posterior part of the tongue—though taste is not lost when the roots of this nerve are affected, as it is probable that the taste fibres of the posterior part of the tongue, soft palate, and anterior pillars pass from the glosso-pharyngeal nerve through its tympanic branch to the small superficial petrosal nerve of the third division of the fifth nerve.

THE PNEUMO-GASTRIC, VAGUS, OR TENTH CRANIAL NERVE.

The vagus and its accessory nerve arise from a longitudinal collection of cells in the medulla opposite the calamus scriptorius, which extends upwards and outwards along the floor of the fourth ventricle; the upper part of this collection is the nucleus for the glosso-pharyngeal nerve, the middle part is for the vagus, and the lower part for its accessory nerve. All these nerves issue from the lateral surface of the medulla, and after passing through the jugular foramen the accessory part joins the vagus. The accessory part supplies the motor fibres to the levator palati—probably through the pharyngeal plexus,—and to the larynx through the inferior or recurrent laryngeal, and through the superior laryngeal branch (crico-thyroid muscle).

Symptoms of involvement on one side of the vagus and accessory nuclei, or of the mixed nerve, are paralysis of the soft palate and of one vocal cord on the same side; the palate on phonation being drawn up, and the raphé being drawn over, to the opposite side, and the vocal cord being immovable. Aneurysm of the arch of the aorta on the left side, or aneurysm of the right subclavian artery, by compressing the inferior or recurrent laryngeal, produces on one side paralysis of all the laryngeal muscles except the crico-thyroid; or if the paralysis is not complete, the posterior crico-arytænoid—the abductor of the vocal cord—is alone paralysed, owing probably to its being the weakest muscle (Semon); bilateral abductor paralysis occurs when both inferior laryngeal nerves are compressed, or when there is disease of both accessory nuclei as in tabes dorsalis. Above the level of the nuclei the paralysis is always bilateral as in the case of the eyes, but it is necessary to have a lesion in the cortical centres in both hemispheres, or in the descending motor tracts of both sides, to produce this bilateral paralysis; both vocal cords are inervated from either cortex (Horsley and Semon), so that if the cortex or motor tract of one side is paralysed the opposite cortex can cause both cords to act; the bilateral paralysis may be complete or only affect the abductors.

In complete paralysis of both abductors the cords are close together, and instead of being separated in inspiration come still closer together, inspiration is difficult and stridulous, but speaking and coughing are possible; the patient is in a very dangerous condition, as slight swelling of the cords may completely block the glottis. In complete paralysis of all the muscles of both cords, they are slightly apart in, what is known as, the cadaveric position, coughing and phonation are impossible, as the glottis cannot be closed, and there is no stridor except on deep inspiration; in total paralysis of one cord, this is motionless, the voice is altered, but it is not absent, as the healthy cord can be brought over to the paralysed one, but not sufficient to close the glottis for coughing.

The symptoms due to lesion of the vagus itself are a slowing of respiration, a quicker or slower action of the heart, and vomiting, but they are not very common.

THE SPINAL ACCESSORY OR ELEVENTH NERVE.

This nerve consists of two parts—the accessory, which joins the vagus, and the spinal, which arises from the cells in the anterior horns of the cervical cord as low down as the level of the fifth cervical root, and pierces the lateral column in several branches, which join to form the nerve. This ascends into the skull cavity through the foramen magnum and passes out through the jugular foramen to supply the sterno-mastoid, which it pierces, and it ends in the trapezius almost at its lower border; it supplies this muscle along with the third and fourth cervical nerves. The spinal accessory and the branches from the cervical nerves have to be divided in some cases of torticollis.

THE HYPOGLOSSAL OR TWELFTH NERVE.

This nerve arises from its nucleus in the floor of the fourth ventricle at its inferior angle and below the calamus scriptorius, lying on the inner side of the vagus nucleus. The nerve lies close to, but anteriorly to, the vagus and spinal accessory nerves, and leaves the cranial cavity by the anterior condyloid foramen, and supplies all the intrinsic muscles of one-half of the tongue, and the stylo-hyoid, hyo-glossus, genio-hyoid, and genio-hyo-glossus muscles, but not the depressors of the hyoid bone, which are supplied by the first and second cervical nerves through the descendens noni.[1]

Paralysis with atrophy of the one-half of the tongue is produced rarely by neuritis of the nerve, as in a case of diabetes under my care, or from pressure on the nerve either in the neck or within the cranial cavity; in this last place it is involved in meningitis especially syphilitic, when it is particularly liable to be associated with paralysis of the soft palate, of one vocal cord, and of the sterno-mastoid and trapezius, all on the same side, due to both parts of the spinal accessory being also involved.

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[1] Horsley and Beevor, *Proc. Roy. Soc.*, 1888.

In paralysis of one-half of the tongue from neuritis, there is flattening with distinct loss of the muscular substance which can be seen and felt, while the mucous membrane is in folds.

The hypoglossal nucleus is especially involved in bulbar cases, and is usually bilaterally affected, so that both sides of the tongue are atrophied, fibrillar tremors are seen, the mucous membrane is in folds, and in extreme cases the organ cannot be protruded beyond the teeth. There is reason to believe that the orbicularis oris is supplied by the hypoglossal nucleus and is paralysed when this is affected, and not when the hypoglossal nerve is affected.

A lesion of the cerebral cortex, where the movements of the tongue are represented, or of the motor tract in the internal capsule, crus cerebri, and pons above the hypoglossal nuclei will produce paralysis of the tongue, so that when protruded it deviates to one side, and the tip cannot be put into the cheek which is on the side opposite to the lesion.

Taste and sensation are not affected by paralysis of the hypoglossal nerve.

CHAPTER XIII.

DISEASES OF THE MEMBRANES OF THE BRAIN.

The most common disease is inflammation, and the symptoms differ accordingly as the dura or the pia mater is the more affected. (For description of cerebral membranes, see p. 99.)

INFLAMMATION OF THE DURA MATER, OR PACHYMENINGITIS.

This is much less common than inflammation of the pia mater, and comprises an *external* form, occurring in the outer part of the membrane which forms the periosteum of the cranial bones, and which is nearly always secondary to injury or diseases of the bones; and an *internal hæmorrhagic pachymeningitis*, affecting both sides of the brain and consisting of layers of fibrinous membranes, with the spaces between the layers filled with blood, in the substance of the dura and between it and the arachnoid membrane. It is also called hæmatoma of the dura mater, and is a rare disease; it occurs mostly in old people, in the insane, and after chronic alcoholism.

Symptoms.—The symptoms of external pachymeningitis are those of the diseased bone which produces the disease; while in the internal form the symptoms are first those of irritation, and then of paralysis of the cortex, and the disease is usually fatal.

INFLAMMATION OF THE PIA MATER.

This is a disease which occurs very frequently, especially in children, and it is found in the forms of simple, purulent, tubercular, and syphilitic meningitis.

Causes.—Heredity is present in the tubercular form; the age most affected is that of childhood, and boys are more attacked than girls. Traumatic causes, as injuries to the head, produce meningitis on the convexity of the brain, especially in adults. Meningitis is also set up by caries or abscess of the nasal or aural cavities, or by an abscess or growth in the brain itself; it is secondary to the acute specific diseases, as measles and septicæmia; and lastly it is caused by definite specific processes, as the tubercular and syphilitic.

Symptoms.—It will be advisable to describe the symptoms of a case of tubercular meningitis, and then to mention how the other forms vary from it. Premonitory symptoms are present, as fretfulness, malaise, vomiting, headache, with some pyrexia for several days. The pronounced symptoms are usually arranged in three stages—the irritative, the paralytic, and the comatose.

The *irritative* stage begins gradually, or sometimes rather suddenly, with severe headache, usually frontal, and heat of the head, causing the patient to cry out; vomiting, which is quite irrespective of food; pyrexia of an irregular type, with as a rule increased pulse and irregular respiration; extreme restlessness and absence of sleep, with grinding of the teeth, the child crying out on being touched; photophobia, severe headache with delirium of a quiet nature or very noisy; the tongue soon becomes furred, and the bowels are constipated. Other symptoms which supervene, due to the cranial nerves being involved, are optic neuritis, sometimes with tubercles in the choroid, especially in basal meningitis; inequality of the pupils, which are usually contracted and do not react properly to light; ptosis, and strabismus; twitching or weakness of one side of the face. Convulsions or rigidity of one-half or of both sides of the body occur; and retraction of the head, especially in basal meningitis. The knee jerk is often absent, due probably to the spinal-cord membranes being involved.

In the *second* or *paralytic stage* the above active symptoms give way to a passive condition; the patient lies in an apathetic condition with some paralysis, with only now and then a shrill cry—the "hydrocephalic cry"—with dilated pupils, irregular pulse and respiration, and lower temperature.

In the *last stage* the child gradually becomes comatose, with dilated, insensitive pupils; general muscular relaxation, with occasional convulsions; involuntary passage of excreta; rapid, irregular pulse; Cheyne-Stokes breathing, with a subnormal temperature or a rapid rise at the end. In other cases the typhoid condition is present, with brown, dry tongue, and picking at the bed-clothes. Death usually occurs from gradual coma or in a fit.

The duration of a case is from one to three weeks.

The above symptoms will differ according to whether the disease affects the base of the brain or the convexity. In the former case individual cranial nerves are involved, and optic neuritis and vomiting are common; while in the latter there are local convulsions of one limb, or a combined movement of the face and both eyes to one side, according to the seat of the irritative lesion in the motor cortex, delirium is present, and vomiting and optic neuritis are rarer.

Of the other forms of meningitis, in the *simple* form the prodromata are often absent, and the symptoms are more irregular. The *purulent* form is often secondary to a suppuration elsewhere, as of the middle ear or the bones of the skull, to injuries, to erysipelas of the face, or to pyæmia and the acute specific fevers; in the purulent cases the temperature has a definite evening rise. The *syphilitic* form is usually more chronic than acute, and consists of a thickening of the membranes, especially the pia and arachnoid; and the symptoms are those of local irritation, followed by paralysis, of the part supplied.

Pathology.—In tubercular meningitis in the earliest stages the membranes, especially the pia, are congested; then they become opaque, and the lymph sheaths along the vessels are distended with fluid, which collects in the meshes of the membranes at the base of the brain and along the Sylvian fissures, it is turbid, but not purulent; fluid also collects in the ventricles, and this is considered to be due to obstruction of the veins of Galen by tubercles. These tubercles are formed later, and occur as minute transparent bodies, the size of a small pin's head or less, in the pia mater. They are found along the course of the vessels, especially at the base of the brain and along the middle meningeal artery in the Sylvian fissure, and also in the choroid plexus of the lateral ventricles, and they consist of lymphoid cells growing around the vessel and within its perivascular sheath, and in them the tubercular bacillus has been found.

In simple meningitis the base of the brain is not especially affected, and the convexity of the hemispheres may be the most involved; the exuded fluid has also more tendency to become purulent, so that the surface of the brain may be covered with pus and the ventricles contain purulent fluid.

Prognosis is most favourable in the simple form due to injury, but even here the condition is often very serious, and may end fatally; the tubercular form is almost always fatal, but a few cases have been recorded in which recovery seems undoubtedly to have taken place.

Diagnosis.—In some cases it is extremely difficult from an absence of definite local symptoms to make a diagnosis. The chief general symptoms are those of an acute inflammatory disease, as irregular pyrexia and pulse, severe headache and delirium occurring together; the local symptoms are due to irritation or paralysis of the cranial nerves, as optic neuritis, unequal pupils, strabismus, or due to cortical irritation as local convulsions. Typhoid and other specific fevers are most likely to be confounded with meningitis, but, as was pointed out by Sir W. Jenner, in typhoid the headache ceases when delirium begins, whereas in meningitis the two coexist. The absence of the knee jerk and the presence of optic neuritis would also distinguish meningitis.

With regard to the different forms of meningitis, if there is a family history of phthisis, and the age is below twenty, the disease is probably tubercular; while with a history of empyema, purulent discharge from the ear, or caries of the temporal bone, the meningitis is probably purulent.

Treatment.—It is of the greatest importance to find any cause for the meningitis; and if there is any purulent disease of the ear, this and the mastoid cells must be treated by surgical means. Any local abscess about the head from caries, etc., must be similarly attended to.

The general treatment consists of absolute rest in a quiet, darkened room, and the diet should consist of light food without stimulants, unless the pulse becomes very weak.

Local treatment, as shaving the head and applying an ice bag or ice water passed through a cap formed of coils of tubing, is required, but strong antiphlogistic measures as blood-letting are not admissible; a blister applied to behind the ears sometimes seems to relieve the headache.

Internally the bowels should be opened by a dose of grey powder or calomel, and afterwards the patient should be kept under the influence of mercury by nightly inunctions in the axillæ of unguentum hydrarg., the gums being carefully watched; the headache is treated by sedatives as the bromides, chloral, hyoscyamus, or antipyrin, vomiting by sucking ice and by the application of a mustard leaf to the epigastrium. In septicæmic meningitis full doses of perchloride of iron have been found useful (Gowers).

CHRONIC MENINGITIS.

This occurs in adults as the alcoholic and the syphilitic forms, of which the latter is the more important. This is found as a thickening of tissue in the pia-arachnoid membranes in the neighbourhood of a syphilitic growth, or it may be quite independent of a growth. The symptoms vary according as the seat of the disease is over the so-called motor area on the convexity of the cortex, or about the cranial nerves at the base; in the former case local tenderness of the skull and localised fits occur, and in the latter paralysis of the cranial nerves as the facial, spinal accessory, and hypoglossal of one side are met with.

In one syphilitic case under my care there was great thickening of the pia arachnoid at the base, causing blindness of the left eye, right hemianopia of the right eye, optic neuritis, and paralysis of the left facial and the right third nerves with at one time partial paralysis of the right fifth nerve. This case was benefited by mercury and iodides for a time, but later they were of no use.

CHRONIC INFANTILE MENINGITIS.

Drs. Gee and Barlow have described in infants this condition, as especially affecting the posterior fossa of the skull. The chronic inflammation sticks together the openings from the fourth ventricle, which becomes distended with fluid and gives rise to spasmodic retraction of the head, rigidity of the limbs, convulsions, with vomiting and some fever; this condition may last for one or several months, and end in death, recovery, or in chronic hydrocephalus. As the disease occurs in syphilitic subjects, the best treatment is rubbing mercury ointment into the back of the neck.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

This disease attacks several people in the same district. It is rare in England, but more common on the Continent.

Causes.—*Hereditary* predisposition is not a factor. The age is usually that of young people below puberty, and it affects males more than females. The disease does not appear to be contagious, and no definite relation can be traced to insanitation or previous ill health, though some cases seem to favour these as causes. It is most prevalent in cold countries, and in cold weather.

Symptoms.—There may be prodromata of ill health and stiffness at the back of the neck, but the onset is sometimes sudden, with vomiting, pain in the head and in the back, followed by rigidity of the neck and back muscles, producing retraction of the head and arching the spine concave backwards. Delirium is added later to the headache, and convulsions may occur. Pyrexia is present, and may be considerable, and the pulse may be increased, or be not more than the normal. Other symptoms are skin eruptions, especially purpura and herpes, the former occurring on the lower part of the limbs, and the latter beginning about the lips; optic neuritis, paralysis of cranial nerves, inequality and dilatation of pupils, loss of hearing, rarely hemiplegia and paraplegia. The disease is also associated with pneumonia.

The course of the case varies. In very acute cases the patient soon becomes comatose, and dies in from a few hours to one or two days; in favourable cases improvement takes place in the second week.

Pathology.—In very rapid cases there may be only slight congestion of the membranes; but in cases which have lasted longer, and of which I had once an opportunity of making a post-mortem examination, there is very intense congestion of the pia mater of the brain and spinal cord, with opacity and exudation of lymph, and sometimes pus in the subarachnoid space; there is also turbid fluid in the ventricles, and the cortex of the brain may have small hæmorrhages or collections of pus. According to most recent authorities, the disease is due to a specific organism which is found in the pia mater and arachnoid, and which is either the organism of pneumonia of Fraenkel or a diplococcus allied to it; this probably produces a poison which causes the symptoms.

Diagnosis from other forms of meningitis is often difficult, and is assisted when there are several cases in the same neighbourhood, and the skin eruption is well, marked and pneumonia is present; from other diseases as small-pox, typhoid, tetanus, and uræmic convulsions the diagnosis is helped by observing that in the first two there is not muscular rigidity, and in the last two there is not usually rise of temperature.

Prognosis is bad, especially if coma ensues in a few days; but the severity of the disease varies in different epidemics.

Treatment is the same as for other forms of meningitis; and as no specific drug is known, the symptoms should be treated.

CHAPTER XIV.

ORGANIC DISEASES OF THE BRAIN.

ANÆMIA OF THE BRAIN.

In a large number of cases anæmia of the brain is a part of a general condition of anæmia, or is brought about by a feeble condition of the heart producing syncope; in others it is produced by pressure of fluid or extravasated blood in the ventricles; tying one carotid produces sometimes local paralysis of the opposite side, while extensive blocking of a large vessel by thrombosis or embolism produces first anæmia and then softening of the part supplied.

Symptoms.—These differ according as the onset is sudden or gradual. In the former the symptoms are those of syncope; the special symptoms attendant to the brain are a drowsy feeling, a dimness of sight, giddiness, noises in the ears, inability to keep the erect posture, pallor of face, cold sweats, with sighing respiration, and weak pulse; in a further stage consciousness is lost with dilated pupils, and general convulsions may occur. In gradual anæmia various subjective sensations of tingling or numbness in the extremities, and headache are found; also black specks before the eyes, noises and beating in the ears, and giddiness. The intellect is dull, but in some cases hallucinations may occur.

Pathology.—Pallor of the brain has been found; but it is very difficult to be sure that this condition existed during life. The condition of unconsciousness, followed by convulsions, would seem to show that the effect of withdrawing the blood supply is first to produce loss of function, and then to decrease the stability of the cortical cells, so that they "discharge" too easily, and convulsions ensue.

Treatment consists in improving the anæmic condition by giving iron and nutritious food. In the condition of acute anæmia, the patient should lie down flat with the head low, and stimulants should be given with local counter-irritation, as a sinapism, applied to the præcordial region and inhalation of ammonia to the nostrils. In the mental derangements, morphia either hypodermically or by the mouth has been recommended.

HYPERÆMIA.

Congestion of the Brain.

Formerly the term was a convenient one for explaining various clinical conditions which occurred without any direct evidence that congestion existed; but now it should be restricted to cases where the symptoms are so transient as to be explained only by a temporary congestion. Two kinds of congestion are described—active and passive.

Causes of active congestion are (1) a condition of general plethora, (2) an increased flow of blood to the head, due to cardiac hypertrophy or excitable action of the heart, (3) contraction of the arterioles elsewhere, and (4) relaxation of the arterioles from nitrite of amyl, etc.; while passive congestion is caused by impeding of the return of blood, as by pressure on the jugulars, or by increasing the intra-thoracic pressure by coughing and straining.

Symptoms.—Active hyperæmia, or what is known as congestive apoplexy, occurs occasionally in elderly plethoric people, who become unconscious with a rise of temperature; this is followed by hemiplegia which is very transient, and only lasts a few hours; while in others there is pain and throbbing of the head, with rise of temperature.

In the *passive* variety there is dull headache, with nocturnal delirium, the face being turgid.

Diagnosis in these cases has to be made with great caution, after excluding on the one side hemiplegia due to a definite lesion, and on the other cases of hypochondriasis. In a transient hemiplegia the diagnosis of congestion is made, when it is hardly possible that a hæmorrhage or a blocking of an artery could produce symptoms of such short duration.

Treatment in passive congestion consists in removing the cause as much as possible. In active congestion, a saline purgative with diuretics is of the greatest importance in relieving the congested vessels, and the same result may be affected by putting the legs into hot water, and locally by venesection from the temporal artery, or leeches to the temples or behind the ears, and ice to the head. At night the patient should sleep with the head well raised with pillows, or in a chair with the legs hanging down; bromides should be given at night, and all causes tending to increase the congestion, as alcohol, rich food, or mental worry, should be carefully avoided.

CHAPTER XV.

ORGANIC DISEASES OF THE BRAIN (*continued*).

VASCULAR CHANGES.

CEREBRAL HÆMORRHAGE, CEREBRAL SOFTENING (THROMBOSIS AND EMBOLISM), APOPLEXY, HEMIPLEGIA.

As the symptoms produced by hæmorrhage from, or blocking, of a cerebral vessel are very similar, and most frequently present themselves in the form of hemiplegia or paralysis of half the body, they will be described together.

Causes.—*Hæmorrhage* occurs either on the surface of the brain, into the membranes, or into the substance of the brain, and it is the latter which is the more common. The vessels are weakened by atheromatous degeneration, and their walls yield at the weak points, and thus miliary aneurysms of the size of from $\frac{1}{25}$th of an inch and less are found in great numbers, and these are liable to give way and produce a hæmorrhage; granular kidney, by causing cardiac hypertrophy and by producing changes in the walls of the vessels, predisposes to hæmorrhage, while gout and alcoholic excess also produce degeneration and weaken the arteries.

Acute *softening* occurs from the blocking of a vessel by thrombosis, or by an embolus lodging in a vessel at its bifurcation.

Thrombosis is caused by—(1) Atheroma of a vessel at the base of the brain, giving rise to a projection into the lumen of the vessel, as a yellow thickening or a calcareous plate, on which a clot can readily form; gout and alcoholic excess predispose degeneration of the arteries and atheroma, and take place especially in old people. (2) Syphilitic arteritis, which occurs in the larger arteries at the base of the brain, consists of a growth between the inner coat and the elastic lamina, which projects into the lumen of the vessel, and thus favours the formation of a clot; it occurs between thirty and forty years, or even as young as twenty. (3) Besides changes in the arteries, some conditions of blood, especially in the puerperal condition, during the course of wasting diseases and in profound anæmia, are very prone to produce coagulation of the blood, and a slow circulation acts in a similar manner.

Embolism is caused by a vegetation from a diseased cardiac valve, or by a piece of clot, detached from a plugged vessel in other parts of the body, being carried into the circulation. Mitral stenosis is the most common cardiac lesion, and a recent endocarditis is more commonly the cause than old-standing disease. The vegetation or piece of clot is carried along by the blood stream, and is usually caught at the bifurcation of an artery, especially of the left middle cerebral; and if the vessel be completely blocked, a clot is formed beyond the obstruction, and on the cardiac side as far as the nearest branch.

Heredity.—A tendency for certain families to have hemiplegia is noticed, and is probably due rather to inherent disease of the vessels than of the nervous elements themselves.

Age.—Hæmorrhage is most common in elderly people after forty; softening from thrombosis occurs after thirty and up to old age; while embolism is most common between youth and middle age.

Exciting causes.—An attack of hæmorrhage is brought on by violent exertion, or straining at stool, or coughing; but this is very seldom the case with embolism, and rarely with thrombosis. An injury to the head may produce a meningeal hæmorrhage.

Symptoms will differ according to the nature, position, and extent of the lesion, and as to whether it is due to a hæmorrhage from, or a blocking of, a vessel. Although vascular lesions may occur in any of the cerebral arteries, the middle cerebral which supplies the internal capsule is so very frequently affected that the symptoms of the lesion, *viz.* hemiplegia, will be here described. These symptoms may be separated into premonitory or transient, and permanent.

The *premonitory* symptoms may occur for some weeks or only for a few hours before an attack, and consist of giddiness, headache, tingling and numbness down one arm and leg, with transient weakness, failure of memory, and irritability; they are most common in thrombosis, uncommon in hæmorrhage, and rare in embolism, and are caused by a partial blocking of an artery, causing temporary slowing of the blood stream.

The *permanent* symptoms in a severe case consist of a sudden loss of consciousness, in which the patient falls to the ground. In this condition all voluntary movements are lost, the limbs are relaxed and drop when raised. There is no failure of the heart's action as in syncope; the respiration is stertorous, with blowing out of the cheeks; the face is flushed, and wet with perspiration, or pale; the pupils are dilated, with slight or no reaction to light; and with regard to the other reflexes, the conjunctival and plantar are lost if the unconsciousness be profound. Swallowing may be impossible, and retention or involuntary passage of urine may exist. Death may occur in a few minutes, or unconsciousness may last for hours or days. Later on, when the coma is less profound, if the lesion be near or in the internal capsule, there are symptoms of paralysis of the arm and leg of one side, which are found to be more flaccid than on the other side, when the limb is lifted up and allowed to fall. In addition there is often conjugate deviation of the eyes (see fig. 4)—*i.e.* both the eyes remain turned away from the paralysed side—and likewise the head is rotated to, and the mouth is drawn towards, the non-paralysed side, due to the over-action of the unaffected muscles. In some cases "early rigidity" occurs in the paralysed limbs, due probably to the irritation of a large lesion. The knee jerks which may have been lost in the stage of profound coma often become increased on the paralysed side. Difficulty of swallowing may be present. The temperature in a severe case, due to hæmorrhage, usually falls in an hour or so after the onset from one to three degrees; and if the case is going to end fatally, either it never rises, or it goes on rising rapidly till the end. When death occurs in a few days, the temperature becomes stationary, and then a rapid rise occurs. In cases which recover, there is a slight rise after the initial fall, and then a return to the normal (Bourneville). There is often a slight increase of temperature on the paralysed side. The most profound unconsciousness and fall of temperature are commonly due to the shock of a hæmorrhage lacerating or compressing the brain substance, or less commonly to the blocking of a large vessel. In other cases due to extensive thrombosis, the mental condition remains drowsy with transient excitement, the patient answering when spoken to, but relapsing at once into a heavy sleep.

After unconsciousness of a few hours or days, the patient recovers his senses, with paralysis of one-half of the body, and if the right side be affected there may be difficulty of speaking and aphasia (see p. 281). In the face, on the paralysed side the naso-labial fold is less pronounced, there is slight difficulty in closing the eyelids of the one side, and inability to retract the angle of the mouth though the power of pursing up the mouth as in whistling is retained, and the tongue when protruded usually goes towards the paralysed side; this is the condition which is usually described, but of more importance to note is the inability to put the tip of the tongue into the cheek of the paralysed side. Rarely the levator palati is paralysed on the same side as the face. The upper and lower limbs may be completely paralysed on one side, so that even the shoulder cannot be shrugged or the hip joint moved. The knee jerk is usually increased, and the wrist tap and ankle clonus are obtained on the paralysed side, while the superficial skin reflexes are diminished. There is no wasting and no alteration to electrical stimulation of the muscles. The sphincters of the bladder and anus are not affected, except when the mental powers are defective, or in cases of double hemiplegia; and it is in these causes that trophic changes as bed sores occur, and especially over the gluteal region of the paralysed side. In severe cases the abdominal muscles may be weaker on the paralysed side. Return of power gradually ensues in severe cases after several weeks, and usually first in the movements of the hip, followed by those of the knee, ankle, and toes. In walking the patient either drags the paralysed foot along the ground or circumducts it; the upper limb begins to recover as a rule after the lower limb, and in the order of shoulder, elbow, hand; the face begins to improve about the same time as the arm. Those joints which do not recover gradually become rigid; the fingers, for instance, becoming flexed into the palm, and the elbow flexed, showing what is called "late rigidity," and at the same time the deep reflexes are much increased. The order of recovery given above was explained by Sir W. Broadbent, by considering that the movements of the two sides of the trunk were so in the habit of acting together, that the muscles of the two sides could be inervated by one cerebral hemisphere alone, and that the movements of the shoulder, being less highly specialised than those of the hand, the former could be brought into action more easily than the latter by the hemisphere of the same side when the opposite hemisphere was paralysed. So that in hemiplegia, according to this theory, the movements which are represented in each hemisphere, as those of the abdominal muscles, recover first, while those of the hand muscles, which are represented in the opposite hemisphere almost entirely, recover last, the muscles of the leg being intermediate. It should be noted that in paralysis of the face in hemiplegia the frontalis muscle usually escapes and the orbicularis palpebrarum is much less affected than

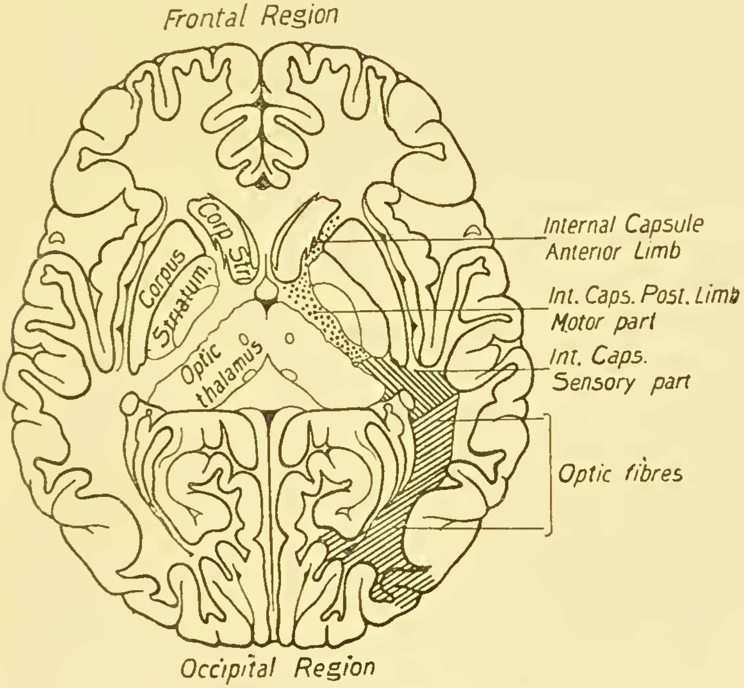

Fig. 24.—Horizontal section (Flechsig) through cerebral hemispheres at the level of the basal ganglia, to show the area of softening in a case of thrombosis, affecting the right internal capsule, motor part slightly, and the sensory part and the optic fibres severely, as shown by the dots and the shading. The symptoms were slight paralysis of left side, with complete left hemianæsthesia and left hemianopia.

in paralysis of the facial nerve, also that the paralysed muscles move equally on the two sides during emotional conditions as laughing.

Sensation, which should always be tested as well as the visual fields, is as a rule not affected; but in a certain number of cases hemianæsthesia or loss of tactile sensation in the paralysed limbs and half the trunk and head of the same side is found from the beginning, and associated with this occasionally hemianopia or loss of the corresponding half of the field of vision in either eye—*i.e.* with right hemiplegia the patient cannot see any object to the right of the middle line with either eye. There may be also loss or deficiency of hearing, taste, and smell (?) on the same side as the anæsthesia (fig. 24).

The above is a description of a typical severe case; in other instances, less severe, there may be only slight or no loss of consciousness, and paralysis of the one side may be not quite complete, and movement is recovered in a few days or weeks. In other cases, although consciousness may not be lost, the resulting hemiplegia may at first be as complete as in the severe cases.

In those cases where consciousness is not lost or only slightly so, the loss of power may have come on suddenly or gradually in one limb, followed in the course of an hour or so by loss in the other limb or the face of the same side; or in other cases the loss of power affects only the face and arm, or the arm and leg. In the unusual cases, where the leg is paralysed more than the arm, the face is either very slightly affected, or it recovers very soon. In attacks which occur during sleep it is often not possible to ascertain whether there was any loss of consciousness.

Hemiplegia may also be caused by hæmorrhage into the meninges, or rarely into the cortex, from the giving way of a miliary aneurysm, when the onset usually begins with convulsions; by hæmorrhage into the crus cerebri or pons Varolii, or by thrombosis of their vessels especially the basilar artery (in this case a lesion of the crus cerebri produces paralysis of the third nerve on the same side as the lesion and a hemiplegia of the opposite face and limbs, while a lesion of the pons causes what is known as crossed paralysis, that is paralysis of the whole of the face on the *same* side as the lesion with paralysis of the arm and leg of the *opposite* side—the reason of this is that the nuclei of these particular nerves, as well as the pyramidal tract above its decussation, are damaged by the lesion); also by hæmorrhage into one lateral ventricle, but owing to the blood making its way into the lateral ventricle of the other side the paralysis frequently becomes general and ends fatally—this last form is called ingravescent, from the manner in which the symptoms become gradually worse.

The subsequent condition of a case of hemiplegia after a vascular lesion is either complete or partial recovery, so that there is some weakness of the face in showing the teeth on one side, recovery of a certain amount of power in the shoulder and elbow with very little or none in the hand, the fingers of which are often rigidly flexed, while in the leg there is a return of power in all the joints except the ankle and toes, so that in walking the foot is dragged along the ground or circumducted. In some cases sensibility is lost on the paralysed side, but in the majority of cases if anæsthesia existed it is recovered from.

Hemianopia, however, persists when the other symptoms have passed off. Of the other cerebral arteries thrombosis of the posterior cerebral would cause opposite hemianæsthesia and hemianopia owing to its supplying the posterior part of the internal capsule and the occipital cortex (fig. 14).

Pathology.—The vascular lesion, the symptom of which is most commonly hemiplegia, occurs most often in the middle cerebral branch of the internal carotid artery, and especially on the left side. The internal carotid artery divides at the base of the brain into the middle and anterior cerebral, and sends also the posterior communicating artery to join the posterior cerebral artery (see p. 43).

The middle cerebral being the continuation of the internal carotid artery, and the left internal carotid springing directly from the highest point of the aortic arch, it can be understood that the pressure of the blood is most felt in the left middle cerebral artery, and also that any vegetations from the cardiac valves are more easily swept into this vessel. The middle cerebral artery winds along the fissure of Sylvius to the outer surface, and in its course it gives off antero-lateral branches, called lenticulo-striate and lenticulo-optic, which penetrate the anterior perforated space at the base of the brain, and supply the basal ganglia and the internal capsule. These branches are very liable to rupture, and if the lenticulo-striate is the one affected, the more anterior motor part of the capsule will be destroyed, and, if the lenticulo-optic, the sensory part of the internal capsule will be especially involved. A hæmorrhage therefore acts by mechanical destruction and laceration of the fibres of the internal capsule, whereas a blocking of a vessel causes cessation of nutritional activity and subsequent softening of the part supplied.

A thrombosis or embolism will give different symptoms according to where the obstruction takes place, whether in the middle cerebral before it gives off the basal branches, or after this; in the former case, all the internal capsule will be deprived of its blood supply in addition to the area of the cortex on the outer surface of the hemisphere (see fig. 15, p. 44).

Hæmorrhage occurs most frequently in the basal ganglia and internal capsule, next into the centrum ovale, and least into the cortex cerebri or cerebellum. The extravasated blood tears up the brain substance, and after death it is found clotted and mixed with *débris* of cerebral substance; later the clot contracts, becomes a brown and at last a yellowish colour, while the connective tissue of the brain substance increases, and forms a sort of cyst wall enclosing the clot.

Thrombosis occurs most often in the large vessels at the base, as the middle and posterior cerebral, and in the basilar from atheroma or syphilitic arteritis. Embolism is most frequent in the middle and posterior cerebrals and the vertebral.

In both cases, if the vessel is completely blocked, there is anæmia of the part of the brain supplied, with a zone of hyperæmia all around it, and some extravasation of blood; the area is of a deep red colour, soft, and consists of broken-down nerve fibres, granules of fat, and granular corpuscles from the fatty degeneration of the nerve cells; later on the colour becomes yellow or even white, producing the different forms of red, yellow, and white softening of the brain; in the last stages the softened area dries up and forms a cicatrix.

Hemiplegia is therefore caused by a lesion in the motor path (fig. 12) from the excitable cortex down to the pyramidal decussation. The fibres are destroyed or compressed by the lesion, and cause loss of voluntary power in the parts of the body to which motor impulses are conveyed by these fibres, and the movements which are represented in one hemisphere only are most affected. The most common place for a lesion is the internal capsule, especially the posterior limb. If the fibres are permanently destroyed, they degenerate below, being cut off from their cells in the cortex, and give rise to sclerosis of the pyramidal tract and the opposite lateral column, which produces the symptoms of late rigidity and increased deep reflexes.

Diagnosis.—The cause of the lesion, whether due to hæmorrhage, thrombosis, or embolism, has to be ascertained as well as the situation of the lesion. In the early stage, if there is insensibility, this has to be diagnosed from other forms of unconsciousness; but for the diagnosis of the different causes of apoplexy, it may be said that profound loss of consciousness after a sudden onset, in which there is stertorous breathing, a fall of temperature, and uneven or sluggish pupils, is more probably due to hæmorrhage about the internal capsule than to extensive thrombosis or embolism. A sudden onset therefore with complete loss of consciousness occurring after forty in a strong, healthy-looking man usually means hæmorrhage ; a gradual or rapid onset with slight or complete loss of consciousness, preceded by prodromata, as headache, numbness, drowsiness, in a person overworked or suffering from a weak heart or wasting disease over thirty, is in favour of thrombosis ; while a sudden onset without loss of consciousness in a patient with disease of the cardiac valves below the age of thirty is probably embolism. An initial fall in temperature does not occur in thrombosis or embolism. Bright's disease with a hypertrophied heart predisposes to hæmorrhage, atheroma to hæmorrhage or thrombosis, a history of syphilis to thrombosis, and of rheumatic fever to embolism.

The resulting hemiplegia, if complete, and if accompanied by loss of speech—aphasia is nearly always due to thrombosis—hemianæsthesia, or hemianopia, is probably due to a thrombosis or embolism, as a hæmorrhage large enough to produce symptoms so extensive would in all likelihood have killed the patient. Cortical hemiplegia is distinguished from disease about the internal capsule by beginning with fits, by the paralysis being less extensive, and by loss of muscular sense and of exact localisation of sensation in the paralysed limbs ; hemiplegia from disease about the pons and medulla is distinguished by the third or seventh cranial nerve being affected on the same side of the lesion, while the limbs are affected on the opposite side.

Tumours growing in the internal capsule or centrum ovale cause hemiplegia ; but the rate of progress is measured by weeks and not hours, and there are usually other signs of a tumour (see "Tumours of the Brain").

Prognosis.—In the unconscious stage the first question is, Will the patient die or recover consciousness? Complete unconsciousness with relaxed limbs, dilated and inactive pupils, abolition or impairment of the superficial and deep reflexes, and a subnormal or a rapidly rising temperature, are very serious symptoms, probably ending in death; and the prognosis is worse if there is any exacerbation of these symptoms, often showing that the hæmorrhage has burst into a ventricle. It must be remembered that the blocking of a large artery like the middle cerebral near its commencement may cause profound coma lasting three or four days, but if there is no alteration of the temperature and the coma is not absolute, recovery of consciousness may occur, whereas if this condition were due to hæmorrhage death would almost certainly ensue. A blocking of the internal carotid or basilar arteries is more dangerous to life than that of the middle cerebral, and is rarely recovered from.

After recovery from unconsciousness, it is impossible to say which limbs will recover; but as a rule the leg recovers before the arm. If there is no return of power before a month after the attack, the prognosis is bad for recovery of movement. Early rigidity, especially if it persists, is unfavourable. As long as a limb continues to improve there is always a chance of still further movement returning; but when the loss of power is followed by late rigidity and marked increase of the tendon reflexes, the limb will almost certainly not recover. It must be remembered that in the act of yawning, an upper limb, which is completely paralysed, may be reflexly elevated, and this often gives the patient the erroneous idea that the limb is recovering power, but this is disproved by the inability of the patient to move it voluntarily. Return of power is more likely to occur after thrombosis or embolism than after hæmorrhage. After lesions causing hemiplegia, hemianæsthesia, and hemianopia, the two former may be recovered from leaving a permanent hemianopia. Hemiplegia due to growths in the centrum ovale or internal capsule is necessarily fatal.

Treatment has to be directed to (1) the apoplectic state, and (2) the hemiplegic condition.

In the early condition, after an attack absolute rest is essential, and the head should not be allowed to be too low.

The mode of treatment differs very much according as the attack is due to hæmorrhage from or blocking of a vessel. In the former case, if there is profound unconsciousness with stertorous breathing and cyanosis, sinapisms and warmth should be applied to the legs, stimulants should be withheld, and a drop of croton oil in a little butter put on the back of the tongue to open the bowels and relieve the cerebral pressure; in extreme cases, especially if the heart is hypertrophied and acting strongly and regularly and the pulse incompressible, and if the urine contains albumen, venesection with the abstraction of eight to ten ounces of blood may be required. To further stop the hæmorrhage a grain of ergotin, 2 to 4 minims of the inject. hypodermica, may be injected, and sinapisms applied to the back of the neck. Attention should be paid to the bladder, and a catheter passed if there is any retention, and the urine examined. In prolonged unconsciousness, if the patient cannot swallow, nutrient enemata are required.

In other cases, where a diagnosis of thrombosis or embolism can be made, and where there is slight or no loss of consciousness, strong purgation and venesection would only do harm ; and if the pulse be weak, and there be any sign of collapse, diffusible stimulants as ether and ammonia are indicated, combined with small doses of digitalis if the heart's action be irregular. When the thrombosis occurs in old people, stimulants are required with warmth to the extremities, and liquid food, especially if there is much wandering at night. In these cases the prodromata should be treated, and thus an attack may be staved off, by keeping the patient in a recumbent posture, the extremities warm, giving food with a little stimulant at frequent intervals and during the night, and administering diuretics as spirits of nitric ether, nitrate of potash, and liq. am. acetatis.

As the treatment for hæmorrhage and that for softening from thrombosis or embolism are so opposite, it is best neither to deplete nor to stimulate unless the diagnosis is fairly certain, and unless the symptoms demand one or the other.

If the attack begin with convulsions the bromides should be given, and if the patient be unconscious they should be given in an enema combined with chloral hydrate.

In the hemiplegic condition due to hæmorrhage, the bowels should be kept open, and the action of the skin and kidneys promoted by diaphoretics and diuretics ; if due to thrombosis from syphilis, iodide of potassium in 5-grain doses three times a day should be given; headache should be treated by bromides or gr. x of antipyrin. At the end of two weeks or a month, according to the severity of the case, quinine, iron, and strychnia should be given.

Local treatment should not be begun too soon, but after from two to four weeks, passive rubbing of the paralysed limbs with lin. camph. should be used, followed by faradisation of the paralysed muscles. Electricity has no effect as far as we know in restoring power to a paralysed limb, but it is well to keep the muscles exercised, so that when the central nervous system recovers, the muscles may be in the best condition to receive the motor impulses.

In prolonged cases bed sores should be guarded against by a water bed, and the passage of urine attended to. The patient should be encouraged to try and move the limbs, and be got into a chair as soon as he is well enough to bear it, and later on he should be encouraged to walk with assistance.

After recovery from an attack, and before an attack in people who have degenerate arteries, attention should be paid to the mode of living, which should be very quiet, with light easily digested food, and the avoidance of excitement or violent exercise.

Later on means should be employed for diminishing the rigidity of the paralysed limbs by straightening the fingers and fixing them in that position, and with the foot the faulty position of extension of the ankle may be guarded against in the early stages by supporting the foot with sand-bags. And when the patient begins to walk, if flexion of the knee is preserved, a boot with a spring to dorso-flex the ankle may be required. Tenotomy is not as a rule necessary. If, notwithstanding this treatment, motor power does not return, it should be remembered that the rigidity is not a cause of the paralysis, and does not act by preventing recovery, but is rather a sequence and the result of the lesion, which produces the paralysis.

INFANTILE HEMIPLEGIA.

Although infantile hemiplegia is due to other changes besides vascular, it will be most convenient to describe it here.

Paralysis of one side occurring in children may be grouped in the following list :—

CONGENITAL.

Cerebral malformations.
Birth paralyses from meningeal hæmorrhage.
  ,, ,, ,, cortical laceration.

ACQUIRED.

Primary.
Secondary to pneumonia.
  ,, ,, scarlet fever, measles.
  ,, ,, bronchitis, whooping-cough.
Traumatic.

In all these conditions the resulting symptoms are paralysis, associated often with convulsions of the paralysed limbs.

CONGENITAL INFANTILE HEMIPLEGIA.

In cerebral malformation all degrees of paralysis are met with, from slight hemiplegia affecting one arm and leg to complete paralysis and rigidity of all four limbs. Convulsions occur in the paralysed limbs in some cases. The head is usually small and narrow across

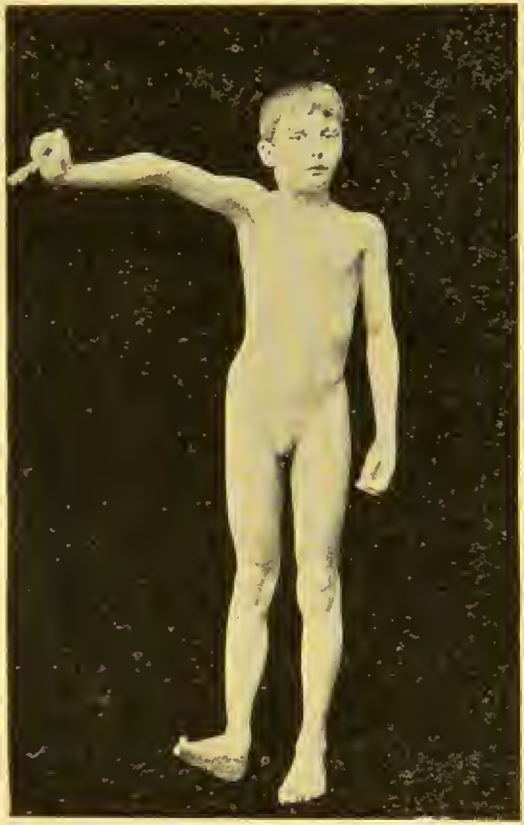

Fig. 25.—Photograph of a case of infantile right hemiplegia, with spasmodic abduction of the humerus, over-extension of the elbow and wrist, and dorso-flexion of the ankle and extension of the big toe. The upper convex border at the elbow represents the anterior surface of this joint, the forearm being pronated.

the forehead (microcephalic), and the mind is deficient, so that in some cases the child is late in walking and talking and in attending to the calls of nature, whilst others are quite idiotic. The cortex cerebri is the seat of some malformation or atrophy. In one of my cases, in which all four limbs were paralysed and affected by general epileptic fits, and the child quite idiotic, the convolutions appeared to be without any definite order, the only fissure or sulcus which could be made out in its proper position being the fissure of Sylvius.

In cerebral birth palsies there is usually a history, of prolonged difficult labour, or of an unusual presentation, producing meningeal hæmorrhage over one or both hemispheres, or laceration of the cortex (McNutt).

The children are born comatose, or have fits immediately after birth, affecting one or both sides with drawing back of the head and difficulty in swallowing. If the child survive, it is found not to move one arm and leg so well as the other; convulsions also occur in the paralysed limbs. In some cases both legs are affected, while the arms have escaped; here the lower limbs are very rigid, with marked spasm of the adductors of the thighs and extensors of the ankles, and with increased knee jerk and ankle clonus. In other cases the limbs may be affected with what is known as athetosis, or involuntary mobile spasm, the hand being most involved. This comes on as the limbs recover voluntary power, and consists of slow, involuntary movements of the hand, which is always changing its position, the fingers being slowly flexed and then hyperextended, or the fingers and thumb are adducted together and then separated (fig. 26). The spasm interferes with voluntary movement, so that the patient has to wait till the spasm has passed off. The face is very rarely affected, except in double athetosis involving both sides of the body. Athetosis of the foot usually shows itself by hyperextension of the big toe. The lesion producing athetosis is probably in the motor region of the cortex, but whether it is due to paralysis or irritation is not known, probably the latter. It is also considered to be caused by a lesion near the optic thalamus at the posterior part of the internal capsule.

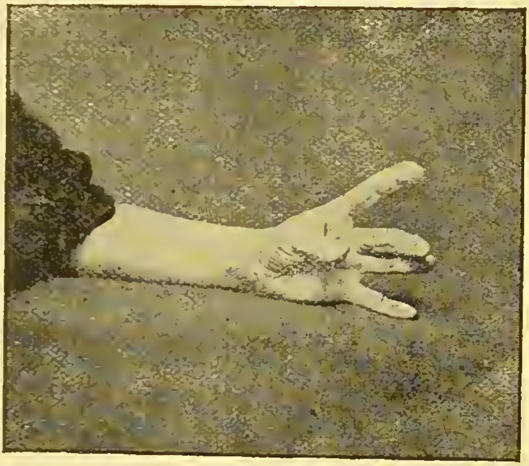

Fig. 26.—Photograph of the left hand from a case of athetosis, showing one of the positions into which the fingers and thumb are constantly changing.

ACQUIRED INFANTILE HEMIPLEGIA.

This differs from the foregoing in coming on after birth, during the first two years of life. In some cases no cause can be assigned ; in others the condition occurs after pneumonia, scarlet fever and measles, or bronchitis and whooping-cough, or after a severe fall. Convulsive fits affecting the whole body or only one side are the first symptoms, and may occur several times for two or three days, and after that the child is found to be paralysed down one side, or the child has one convulsion, and lies in an unconscious condition for several days. The arm and leg are usually paralysed, less commonly the face and rarely speech is affected, so that a child who could say a few words loses all speech. Sensation as a rule is not impaired. Later on a certain amount of power is regained in the leg more than the arm, the joints of which are spasmodically contracted (fig. 25), or affected by athetosis (see above). The growth is retarded, so that after some years the whole hand and arm are uniformly smaller than on the sound side; the knee jerk is excessive, and ankle clonus is present on the paralysed side. It must be remembered that these cases are liable to epileptic fits, involving especially the paralysed side.

The changes causing this hemiplegia have been referred to plugging of a cortical vessel by embolism or thrombosis of a sinus, or to thrombosis of a vein on the surface of the cortex (Gowers), while others have thought that it is due to an inflammation of the cortical grey matter (Strümpell).

Diagnosis has to be made from infantile spinal paralysis (see p. 187), and in the early stage the acquired form to be distinguished from acute meningitis.

Prognosis as regards recovery of power depends on the extent of the lesion, severe cases, and those affected with athetosis, but slighter cases in which some voluntary power is present tend to improve as the child gets older.

Treatment.—In congenital cases, in the early stages, the general health should be kept up by phosphate of iron and cod-liver oil, and small doses of bromide of potassium or ammonium gr. iij to v two or three times a day if there are any convulsions; later on, when the child grows up to five or six years old, it should be specially taught along with other children similarly affected at one of the private homes or public institutions.

In the acquired cases during the attack the child should be kept absolutely quiet, and if it can swallow, bromides in gr. v to x doses every three or four hours should be given while the fits are occurring, or gr. v of chloral hydrate per anum if the child is in a condition of status epilepticus.

Later on the local treatment is the same as that for hemiplegia (*q.v.*), and the child should be encouraged to use the limbs. Deformities, as talipes equinus, are apt to occur, but an operation as tenotomy is less satisfactory than in deformities after infantile spinal paralysis, as in the former they are due to active spasm of the muscles, and not to the unopposed contraction of healthy muscles as in the latter. If the fits are very frequent, and do not yield to the bromides, and begin with a localised aura, it would be quite right to explore the cortex surgically with a view to removing any irritating cicatrix, etc. Athetosis cannot be prevented, and in most cases is permanent, but in one case which I had with Mr. Horsley, due to traumatism, all the cortical movements ceased when the arm centre was removed; but as the operation necessarily produced paralysis of the limb, it should not be undertaken unless the movements are very intolerant.

CHAPTER XVI.

ORGANIC DISEASES OF THE BRAIN (*continued*).

INFLAMMATION OF THE BRAIN.

Abscess.

Inflammation of the brain was formerly confused with softening, which, as we saw in the last chapter, is due to thrombosis or embolism of a vessel; true inflammation is very rarely primary, but is secondary to (1) injury, as fracture or punctured wounds of the skull; (2) disease of the bones, especially caries of the temporal bone following otitis media with suppuration of the middle ear; (3) septic infection by the lymphatics or vessels from pus in the mastoid cells or elsewhere, or from general pyæmia, bronchiectasis, and gangrene of the lung.

Symptoms.—Abscess occurs most in males and young adults. Two different varieties of symptoms exist, the acute and the chronic. In the former, which occurs after an injury to the head such as the kick of a horse, there is severe pain at the place with tenderness, vomiting, pyrexia, rigors, with sometimes optic neuritis, subnormal temperature, and slow pulse, and if the injury is over the motor cortex, localised fits with paralysis; this form, if unrelieved, ends in stupor and death in from a week to a month. These symptoms are found also with pyæmic abscess.

In the chronic form, which commonly follows otitis media, there has been a discharge from the ear for some years, and during this time an abscess has formed, beginning at first with such slight acute symptoms that they have been overlooked. The abscess becomes encapsuled and latent, and gives very little evidence of its presence, except that the patient may have headache at times, and occasional general epileptic fits, while mental changes are observed, the patient becoming more irritable in temper or melancholic. This latent condition may continue for from a month or two to a year or more, and then the abscess may increase and give rise to the symptoms of acute abscess,—increased headache, with tenderness over the lateral or posterior part of the head, one or two rigors, a slow pulse, a subnormal temperature, daily vomiting, and optic neuritis; later on there ensues mental dulness and torpor, ending in coma and death. The above conditions are produced by the pressure of the abscess on the brain substance, and probably the subnormal temperature and slow pulse would not occur if there were an external opening in the cranium, as in a compound fracture.

The two chief sites where abscesses, secondary to otitis media, form are in the first or second temporo-sphenoidal lobe, or in the cerebellum, especially the anterior part of the lateral lobe. The differential diagnosis is often very difficult, but in the case of the left temporo-sphenoidal lobe there might be deafness of the *opposite* right ear, with inability to understand spoken orders, owing to the centre for hearing words in the first temporo-sphenoidal convolution of the left side being involved, or there might be paralysis of the face and arm of the opposite side owing to pressure on the motor cortex.

With cerebellar abscess the above special symptoms are absent, the headache and tenderness are referred more to the occipital region (though this cannot entirely be relied upon), there are vertigo and a staggering gait, with nystagmus, *i.e.* jerking of the eyes to one side.

Another point is the amount of deafness of the affected ear. If hearing for a watch is lost, but that for a tuning-fork placed on the mastoid process is retained, it would show that the disease was probably in the middle ear, with caries of the roof of the tympanum, which lies just beneath the temporo-sphenoidal lobe, where the abscess would probably be found; whereas a tuning-fork not heard on the mastoid process would show that the internal ear was disorganised, and consequently pus would not have much difficulty to pass through the internal auditory meatus, and produce an abscess in the lateral lobe of the cerebellum (Ballance).

Another condition which is intimately connected with purulent discharge from the ear is thrombosis of the lateral sinus. It will be remembered that the lateral sinus, on its way down to the jugular foramen along the inner surface of the skull, has a direction forwards and downwards immediately behind the petrous part of the temporal bone; and if the posterior wall of the tympanum is carious, pus can easily get from the middle ear to the groove for the lateral sinus and thus produce thrombosis, and if the pus be septic, pyæmia is frequently the result. The symptoms are as follows (Ballance):—the presence of purulent discharge from the ear, which may be quite *free* and *inodorous*, or no discharge present; a *sudden* onset with severe pain in the ear and a rigor, followed by headache, a temperature oscillating every twenty-four hours, daily vomiting and repeated rigors, local œdema and tenderness over the mastoid, over the internal jugular vein, or over the lateral sinus, rigidity of the muscles of the back of the neck, and optic neuritis.

Diagnosis.—In cases where there is a distinct history of a blow, followed by tenderness of the part, and with a drowsy condition, a subnormal temperature, and slow pulse, the diagnosis is not so difficult. In other cases it is often very obscure, and especially with regard to abscess about the temporal bone. But given a case in which there is a purulent discharge from the ear, and in which headache, gradual mental dulness passing on to coma, subnormal temperature, and slow pulse ensue, there is very strong evidence that an abscess is forming, and the question as to whether it is in the temporo-sphenoidal lobe or the cerebellum depends on the presence or absence of the symptoms given above. In lateral sinus thrombosis there is headache, but less mental dulness, the temperature is high and oscillates, coming down almost to normal in the morning, the rigors are more frequent than in abscess, and the pulse is quick. Daily vomiting and optic neuritis may occur in either, and show that there is intra-cranial inflammation or pressure.

Optic neuritis, if present, is against the disease being typhoid fever, which, especially when complicated by otitis media, may be confounded with cerebral abscess.

From meningitis the diagnosis is made more difficult by the frequent combination of meningitis with abscess.

In both there may be headache, vomiting, and optic neuritis; but in meningitis there is not the subnormal temperature of abscess or the oscillating temperature of lateral sinus thrombosis.

Prognosis.—Unless a case of acute cerebral abscess is diagnosed early and the pus evacuated, the prognosis is very unfavourable, death being caused by exhaustion or bursting of the abscess into the membranes or into a ventricle.

Treatment.—When the symptoms point to the presence of an abscess, the only treatment is to evacuate it as soon as possible. This is done by trephining with strict antiseptic precautions over the spot where the abscess has been diagnosed to be. The place to operate is shown in many cases by the position of a wound or by tenderness following a blow; but in the case of abscess following otitis media there may be nothing externally to indicate whether the abscess is in the temporo-sphenoidal lobe, in the cerebellum, or whether it is a case of lateral sinus thrombosis.

In recent years great advances have been made in the surgery of these cases, especially by Macewen, Barker, Horsley, and Ballance. The chief guides to reach the various points where abscesses may collect are as follows (Ballance):—

Locality.	Measurements of centre of trephine from centre of meatus externus.	Size of trephine.
Anterior surface of petrous bone	7/8 in. vertically above.	1/2 in.
Mastoid antrum	1/2 " behind, 1/4 in. above.	1/4 "
Lateral sinus	1 " " 1/4 " "	5/8 "
Temporo-sphenoidal abscess	1 1/4 " " 1 1/4 " "	1/2 "
Cerebellar abscess	1 1/2 " " 1/4 " below.	1/2 "

The above measurements are made along the horizontal base line, drawn backwards through the middle of the meatus auditorius externus from the lower margin of the orbit in front (Reid); the last column gives the size of the trephine to use.

When in doubt as to where the lesion is in purulent otitis media, it is best to trephine over the mastoid, removing any pus and gouging away carious bone, so as to expose the lateral sinus; and if this is found to be clotted, to ligature the internal jugular vein in the neck below the clot (Horsley), to cut through the vein above the ligature, and then to slit open the lateral sinus and remove the clot.[1] If in addition there is reason to suspect abscess in the temporo-sphenoidal lobe or cerebellum, the opening in the skull can be enlarged upwards or downwards to reach the spot indicated in the above table.

[1] Cf. Ballance, *Med. Soc. Trans.*, vol. xiii.

CHAPTER XVII.

ORGANIC DISEASES OF THE BRAIN (*continued*).

INTRA-CRANIAL TUMOURS.

Etiology.—Tumours of the brain are more common in males than in females, and in childhood than in advanced age.

Character and situation.—According to the analysis of the fifty-four cases shown at the Pathological Society,[1] the sarcomata were most numerous, next the gliomata and tubercular growths, while psammomata, gummata, carcinomata, fibroid tumours and cysts occurred in the order given; they were found most frequently in the cortex and subjacent white matter, the pons and cerebellum, and less frequently in the central ganglia, the cerebral membranes, and the cranial nerves.

The sarcomata grow from the membranes, the bones, or in the brain substance; gliomata are found in the cerebral and cerebellar hemispheres, and also in the pons; tubercular growths spring from the lymphatic sheaths, especially on the cortex; and gummata from the perivascular sheaths in the cortex.

Gliomata and carcinomata infiltrate the tissues, sarcomata, tubercular, and syphilitic growths do not; and while sarcomata, syphilitic, and tuberculous growths are multiple, the gliomata are not. Rarer forms of tumours are psammomata containing calcareous particles of "brain sand," fibroids, cysts, myxomata, hydatids, and osteomata.

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[1] *Path. Trans.*, 1886.

Symptoms.—These may be divided into two groups —(1) general, and (2) special or localising.

With very few exceptions the onset of the symptoms is gradual.

(1) The general symptoms give a strong indication that there is an intra-cranial growth, but do not help us to localise its position ; these symptoms are headache, persistent vomiting, double optic neuritis, and mental failure.

Headache is usually severe, and may be felt all over the head. Although the pain may not correspond in position to the growth, yet in many cases the pain is in the front of the head with tumours in the centrum ovale, and in the occipital region with cerebellar growths. But if the growth is in the cortex, pain may be localised to one particular spot, and tenderness may be elicited by percussion there. The headache may be constant, with exacerbations which are often intolerable, and the character varies, being dull or acute.

Vomiting occurring daily, of a purposeless character, *i.e.* not depending on the presence of food in the stomach and unattended with nausea, is found most frequently with tumours about the medulla and cerebellar middle lobe, and less commonly with tumours of the cerebral cortex.

Double optic neuritis, which may exist without any affection of sight (Hughlings Jackson), may begin in one eye on the same side or the opposite side to the growth, and is due either to extension of tissue irritation along the optic nerve, to meningitis travelling down the optic nerve to the optic disc, or to fluid distension of the sheath of the optic nerve. Intra-cranial pressure evidently has something to do with producing optic neuritis from the rapid way in which the neuritis subsides in some cases after simple trephining. Optic neuritis is more likely to be caused by a rapid than by a slow-growing tumour, and the quick onset of the neuritis and the degree of swelling are indications of the rate of growth rather than of the size of the tumour. If unrelieved, the neuritis passes on in a few weeks or months to secondary optic atrophy, with defective sight or complete blindness.

Alterations in mental condition may occur from tumours in any part, but especially in the frontal region and the centrum ovale or the corpus callosum, under the form of loss of memory, mental hebetude, depression, delusions, or dementia.

(2) The special or localising symptoms will best be described by taking different regions of the brain, and describing the symptoms which would lead one to diagnose a growth there. The chief regions to be described are the cortex cerebri, the centrum ovale, the internal capsule with the basal ganglia, the cerebellum, and the base of the brain including the anterior fossa, the middle fossa, and the posterior fossa with the crura, pons, and medulla.

The cotrex cerebri.—The symptoms differ according to the part affected, and are (1) those of irritation as convulsions, followed by (2) paralysis of the functions of the part previously convulsed. If the so-called motor cortex is involved (see p. 22), there will be symptoms of irritation as fits, of which the aura, or the first movement, is referred to, or takes place in, the part of the body represented in the part of the cortex affected —*e.g.* if the growth is situated at the upper end of the Rolandic fissure, the first sensation or movement felt will probably be in the opposite big toe. The fits are usually local without loss of consciousness (Jacksonian), or beginning locally may become general with unconsciousness. These Jacksonian fits may occur as many as a hundred a day, and the patient may understand what is said to him, and be able to answer questions while the convulsion is occurring. They begin usually with a feeling of numbness, pain, or tingling in one part of the limb, as the big toe, and directly after with a tonic contraction of the affected part, followed by clonic jerking, which gradually spreads to other parts, following as a rule the arrangement of the centres in the cortex given on p. 22, so that a fit beginning in the big toe would gradually spread up the leg and down the arm and affect the face last. These attacks are followed by paralysis of the convulsed part, and in the case of the limbs increase of the so-called tendon reflexes. These symptoms completely pass off after a few days, unless there is actual destruction of the cortex, when the paralysis is permanent, and it is attended by imperfect localisation for tactile impressions and loss of muscular sense in the paralysed parts (see p. 26). In tumours of the anterior frontal cortex the fits, if present, would probably be those of petit-mal or general epilepsy, there would be no paralysis, but there might be mental hebetude; while a tumour in the left third frontal convolution would be associated with aphasia (p. 281). Of the rest of the cortex localising symptoms could only be obtained from the parts whose functions are more or less known, either in the form of fits or paralysis. For instance, a tumour in the occipital region, in the first temporo-sphenoidal convolution, or in the tip of this lobe may produce fits preceded by a visual, auditory, or an olfactory aura respectively, such as flashes of light, ringing of bells, or an unpleasant smell (a symptom, which preceded attacks of petit-mal, in a case of tumour of the tip of temporo-sphenoidal convolutions in a case I published with Dr. Jackson); later on there may be paralysis of the functions of these parts causing permanent hemianopia or deafness of the opposite side—or word deafness if the lesion is on the left side; while one in the angular or supra-marginal convolution would probably cause some alteration in the field of vision, or, if on the left side, a form of aphasia called word blindness. Occasionally headache is most marked, and there is tenderness to percussion of the skull, over the seat of tumour.

The *corona radiata* and the *internal capsule* with the *basal ganglia* may be the seat of tumours, which are characterised by a motor hemiplegia of the opposite side of a gradual onset, taking weeks to develop, and attacking one part of the body, as the arm, and then gradually involving the leg or face, but as a rule without any fits. If fits occur, the growth is more likely to be in the corona radiata than in the internal capsule, while sensation affecting the half of the body is more likely to be involved by a tumour in the latter part.

Cerebellar tumours.—The localising symptoms differ according as the tumour involves the middle lobe, the middle peduncle, or the lateral lobe (see p. 41). In the case of the middle lobe, the symptoms are a staggering reeling gait, with a tendency to fall forwards, backwards, or to one side; the staggering movements are larger in character than in locomotor ataxia, and the patient lurches bodily to one side more like a drunken man or a landsman on a rolling ship. As a rule there is no definite paralysis unless the pyramidal tract in the pons is pressed upon. A feeling of giddiness is sometimes present, and nystagmus—a jerking of the eyes on looking away from the middle line. The knee jerk has been found absent on both sides in some cases, but as a rule it is increased. Curious fits, with tonic spasm of the muscles of the back, and arching of the spine are described (Hughlings Jackson), but it is doubtful whether they are not caused by pressure on the pons and medulla. In cerebellar tumours it is often difficult to tell which way the patient does fall; it is therefore best to test the walking every day and note each time which way he does fall.

Tumours pressing on the middle peduncle are very rare, and give rise to rolling of the patient round his long axis; while if one lateral lobe be alone involved, it is said that no direct localising symptoms are observed (Nothnagel), unless there is pressure on the pons, which may give rise to paralysis of the limbs on the opposite side; but I have had one case under my care in which pressure on one lateral lobe caused giddiness and staggering to the opposite side, and another case with the same symptoms where the growth was successfully removed (Lunn and Ballance).

A tumour of the corpora quadrigemina produces paralysis of the associated movements in both eyes, such as looking upwards.

At the base of the brain tumours in the *posterior fossa* below the tentorium are similar to those inside the medulla and pons, except that in the former case the cranial nerves, as the sixth, seventh and eighth, are paralysed, and later on motion is gradually lost in the opposite limbs; or the spinal accessory and the hypoglossal nerves are involved, causing paralysis of half the soft palate, half the tongue, one vocal cord, and the trapezius and sterno-mastoid all on the same side; while with tumours, such as glioma or tubercular growths, inside the pons and medulla, there is often facial paralysis on the side of lesion, with conjugate deviation of both eyes *away* from the side of the lesion, due to the involvement of the sixth nucleus which supplies the opposite internal rectus as well as the external rectus of its own side. Paralysis of the sixth nerve and of the external rectus alone is of little localising value, as owing to its long course it is easily affected by pressure under the tentorium cerebelli. Tumours of the *middle fossa* are characterised by a lesion of one fifth nerve, producing paralysis of the muscles of mastication, anæsthesia of the face, and trophic inflammation of one eye on the same side as the growth; and sometimes, from involving the crus cerebri, by paralysis of the third nerve of the same side with loss of power of the opposite face and limbs. In the *anterior fossa* there may be loss of smell of the same side as the growth, with paralysis of the nerves entering the orbit, loss of sight, and paralysis of the muscles of one eye, of that side.

Diagnosis.—In some cases the diagnosis of tumour is easily made; in other cases it is impossible from want of symptoms.

Taking the general symptoms first—*viz.* headache, daily vomiting, and double optic neuritis—the two diseases, in which these symptoms are also found, are extreme anæmia and Bright's disease; but the pallor in the first case and the rapidity with which the symptoms usually disappear when treated by iron, and the presence of albuminuria with a high tension pulse and degenerative changes in the retina, would exclude these diseases.

In the absence of the general symptoms, the special localising symptoms may assist in making a diagnosis. In the case of a tumour of the cortex, it may be very difficult, in the absence of optic neuritis and headache, to say whether frequent fits, which always begin in the same place (*e.g.* the thumb or big toe) and affect one side chiefly, are due to a tumour or not. If there is no loss of consciousness in the attacks, and if they are followed by temporary paralysis of the part convulsed, there is strong evidence in favour of a cortical lesion of a gross character, probably a tumour, and this evidence is much strengthened if there is permanent paralysis with cortical anæsthesia between the fits. On the other hand, a cortical tumour may cause fits, beginning locally but with loss of consciousness like ordinary epilepsy; here a tumour can only be suspected in the absence of the general symptoms, if the fits are very frequent, or if there is permanent paralysis of the part convulsed.

When compared with the paralysis due to vascular lesions as hæmorrhage or thrombosis, the hemiplegia due to a tumour in the corona radiata or internal capsule has nearly always a slow, gradual onset, taking weeks to develop and attacking successively the limbs and face.

In cases where the general symptoms are present, a probable diagnosis can sometimes be made only by the process of exclusion; absence of fits and paralysis would exclude the motor cortex and the internal capsule and pyramidal tract, exemption of the cranial nerves would exclude the base of the brain, and the absence of visual or auditory symptoms would exclude the occipital or temporo-sphenoidal lobes, thus leaving the frontal or cerebellar regions, between which places it is often most difficult to decide.

If a growth is diagnosed, its position may be recognised by the symptoms already described, while its nature can only be surmised from the character of the growths most frequently found in that position, and also taking into account the presence of tumours elsewhere in the body, and the occurrence of a family or personal history of tuberculosis, syphilis, or cancer.

Diagnosis has to be made from acute meningitis and abscess, in which the general symptoms resemble those of tumour; but in the first disease the symptoms are more acute with pyrexia, while abscess is usually associated with some injury or with otitis media, and the symptoms when they develop are more acute, while the temperature is often subnormal.

Finally, it must be remembered that a very large number of patients, suffering from nerve exhaustion, think that they have a cerebral tumour, and the diagnosis can only be made by carefully looking for and excluding all symptoms of tumour.

Prognosis.—This varies with the nature of the growth. Syphilitic tumours are usually, but not always, amenable to treatment; tubercular growths may become dormant; while the termination in other cases is death, unless relief can be given surgically. The end is usually gradual, but some cases die suddenly, especially tumours in or pressing on the medulla, or cerebellar cases.

Treatment.—In cases where a syphilitic history can be made out, mercury, either by rubbing ung. hydrarg. into the axillæ every night until the gums begin to be touched, or by liq. hyd. bichlor., followed by iodide of potassium, beginning with gr. v, and rapidly pushing it to gr. xxx, three times a day, should be employed.

Tubercular growths should be treated with cod-liver oil, maltine, and syrup of the iodide of iron. If the nature of the growth cannot be ascertained, it is best to treat the case as syphilitic.

In any case, after the treatment has been persevered in for from six weeks to two months, if there is no improvement, but advancing of the symptoms, the advisability of an operation should be considered. Thanks to the labours of Macewen, Godlee, and Horsley, cerebral tumours can be removed with safety, provided that strict antiseptic precautions are used. An operation may be required either to remove a tumour, or failing that to relieve the symptoms. The tumours which can most easily be removed are those which grow from the membranes and press on the so-called motor cortex, and next those which are growing in the white matter just below the cortex, both of which would produce localised fits. The position of the trephine hole would be guided by ascertaining the place of representation in the cortex of the part of the body, which is first affected (tingling or spasm) in the fits (see figs. 8 and 9, p. 20). The most important landmarks to find are the relations to the skull of the fissures of Rolando and of Sylvius.

The upper end of the fissure of Rolando is found by measuring along the middle line of the head, which should be completely shaved, from the root of the nose to the occipital protuberance, and half an inch behind the middle point of this distance gives the upper end of the fissure (Thane), which runs forwards and downwards making an angle of 67° with the middle line; it is bounded below by the fissure of Sylvius, which it does not quite reach.

The fissure of Sylvius is found by drawing a line on the shaven scalp from the notch at the junction of the malar with the frontal bone to a point three-quarters of an inch below the parietal eminence, and the posterior three-fifths of this line gives the horizontal limb of the fissure which is at the level of the top of the parieto-squamous suture, while the anterior vertical limb extends upwards from this line from a point situated over the middle of the zygoma (Reid). From the fissure of Rolando the ascending frontal and parietal convolutions can be found. For more detailed account for finding these and other convolutions, other works[1] on the subject must be consulted, and also treatises on surgery for the best mode of performing the necessary operations.

The tumour should if possible be removed; but if it is too large to do so completely, it is best to leave it alone; the mere release of the pressure, which is usually well shown by the bulging of the brain into the trephine hole, relieves the intense headache, the vomiting, the number of the fits, and causes a cessation of the optic neuritis. So that in all cases where there is evident pressure, and even when a tumour cannot be localised, it is advisable to trephine over the seat of greatest pain. In the case of the cerebellum where it is often impossible to tell with our present knowledge in which lateral lobe the growth is, or if in the median lobe to which side it inclines, it is best to trephine over the cerebellum on the side where there is most pain.

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[1] Reid, *Lancet*, 1884; Horsley, *Internat. Jour. Med. Sc.*, April, 1887; Quain's *Anatomy*, tenth edition, vol. iii., last part.

There is one class of cases in which the advisability of removal of a tumour should be carefully considered, and that is in which a tumour grows in the left side of the cortex near the lower end of the fissure of Rolando, as any attempt at removal, unless the tumour was growing from the membranes and only indenting the cortex, would be followed by aphasia.

In cases where an operation cannot be carried out, or does not relieve the symptoms, pain should be relieved by sedatives as chloral, bromides, cannabis Indica, antipyrin, or by morphia, while counter-irritants or ice to the back of the neck are useful.

Intra-cranial Aneurysm.

Like arteries of other parts of the body, the large cerebral arteries are liable to aneurysm.

Causes.—Males are more affected than females, but they apparently occur in young adults as frequently as in the middle-aged.

The direct causes are syphilitic disease of an artery; embolism of an artery, especially by septic material, leading to dilatation of the vessel on the side nearest to the heart; injuries to the head; and a primary degeneration of the arteries in elderly people.

Situation.—The middle cerebral artery and the basilar are most frequently involved, and next to them the internal carotid and the anterior cerebral. The aneurysms are usually sacculated and round in shape, and they compress or destroy the neighbouring cerebral tissue or bones.

Symptoms are often very dubious, and may be absent; when present, they begin gradually, and are general and local. The general symptoms are headache, mental dulness, irritability, and double optic neuritis, which may occur late in the disease. The patient may experience a pulsation, and may hear a murmur, which in some cases can also be heard by auscultating over the skull. In such cases, localisation is assisted by observing if the sounds cease by compressing either carotid, or the vertebrals in the suboccipital space (Grant).

Local symptoms are such as would be caused by pressure on particular parts of the brain and on certain cranial nerves, such as the second, third, fourth, and sixth, which are liable to be involved by an aneurysm of the internal carotid artery, producing loss of sight of one eye and paralysis of its muscles; or if on the left middle cerebral artery, by compressing the under surface of the left frontal lobe and the internal capsule, an aneurysm may produce a gradual hemiplegia with aphasia, as in a case which was under my care; or if on the basilar artery, pressure on the pons or medulla and a cranial nerve, as the fifth or seventh, may cause gradual paralysis of the limbs on one or both sides, and of the cranial nerve involved, together with occipital headache vomiting and sometimes giddiness.

Intra-cranial aneurysms may become obliterated by clotting, or may kill the patient by bursting or by pressure on the surrounding brain tissue. When the aneurysm bursts, the patient quickly becomes comatose, with convulsions if the cortex be irritated, and death occurs rapidly; death from pressure is more gradual and is similar to that of any other tumour.

Diagnosis from any other form of growth is impossible, unless a murmur can be heard; but in some parts, as the neighbourhood of the cavernous sinus, as an aneurysm is more common than other forms of tumours, its presence may be suspected.

The diagnosis of the different arteries affected is sufficiently indicated by their symptoms already given.

Prognosis is very grave, for though some aneurysms have become cured, a fatal termination occurs in most cases, though it may be delayed two or three years.

Treatment.—The general treatment is rest and the avoidance of rapid movements and of allowing the head to hang down; while to promote the formation of a clot, iodide of potassium up to gr. xx or more, three times a day is useful; or the method proposed by Tufnell of giving the minimum of food and fluids with complete rest. In aneurysm of the internal carotid or the middle cerebral arteries, ligature of the common carotid of the same side is indicated, and for a basilar aneurysm ligature of one or both vertebrals.

CHAPTER XVIII.

ORGANIC DISEASES OF THE BRAIN

(*continued*).

DEGENERATION OF THE BRAIN.

Disseminated Sclerosis.

This disease (which is also called multiple or insular sclerosis) is characterised by symptoms having a gradual onset, and it is produced by patches of sclerosed tissue scattered about the central nervous system.

Three forms of the disease are described, cerebral, spinal, and cerebro-spinal, according to the part involved; but it will be sufficient if the last named is described, as it is the most common.

Causes.—*Heredity.* There is often no definite neurotic history in other members of the family, or there is a history of epilepsy or "general nervousness." In one family I have seen, a brother and sister were affected with the disease.

The age of the disease is that of young adult life, from puberty up to thirty or older, and women are more frequently attacked than men. Of exciting causes, mental worry, anxiety, and overwork, together with cold and exposure to wet, seem to be most conducive. Syphilis does not seem to play any part in causation.

Symptoms.—The onset is gradual, and sometimes very insidious, and indefinite symptoms may supervene and pass off for some years before the disease can definitely be recognised. These preliminary symptoms have often been taken for manifestations of hysteria (Buzzard), from which it is hardly possible to distinguish them—in fact I think it highly probable that they may be hysterical and therefore precede on the functional side the breakdown on the structural side, which subsequently occurs—and consist of transient numbness and loss of power in the limbs, temporary diplopia, and loss of sight, from which the patient recovers apparently completely; later on there is some difficulty of walking, and in time the case is fully marked.

In a typical case the chief symptoms are the peculiar tremors and speech, nystagmus, the difficult gait, an increase of the tendon reflexes with some rigidity of the legs and optic atrophy.

The *speech* is described as being slow and scanning, each syllable being spoken slowly and disjointed from the next one; but in other cases each syllable is separated, and is blurted out in such an explosive manner that the words cannot be understood.

The *tremor* does not exist when the patient is lying down and is completely absent when he is not making any muscular effort; but on trying to take hold of an object or to touch the tip of his nose, to and fro movements of the hand and arm ensue, which become larger and more violent as the object is approached. A favourite way of showing this symptom is to make the patient take up a glass full of water and drink it, when the movements become very violent and the water is spilt as the mouth is approached. As a rule the tremor is not increased by closing the patient's eye. Writing in the same way is almost impossible, the pen being dashed about the paper; but in the early cases the writing is merely tremulous. In extreme cases the head shakes, nodding to and fro when the patient sits up in bed, and also tremor of the tongue occurs when protruded. The rate of tremor is not very quick, being about from 3 to 4 oscillations per second.

Nystagmus—the name given to the tremulous movement of the eyes—is not seen when the eyes are at rest looking forwards; but on extreme looking to a fixed point on one side rapid, jerking, parallel movements of both eyes towards that side are made with a slow swing back, the movement being most in the eye which is nearest to the side looked at. Nystagmus is also seen on looking up or down, though less frequently. Later on nystagmus occurs on merely fixing the eyes on an object in front. Sometimes the power of turning both eyes to one side is lost; and rarely there is paralysis of a single nerve, as the third.

The *gait* may be only a little unsteady, or it may be distinctly tremulous, in which case each leg, as it is advanced and the weight of the body put upon it, trembles; or the walk is that due to lateral sclerosis of the cord, dragging the feet along the ground with a side-to-side movement of the body.

Associated with these tremulous movements of the limbs there is gradual general weakness, but no paralysis of any definite movements. The lower limbs as a rule tend to become rigid, the knee jerks are increased, and ankle clonus can be obtained.

Cutaneous sensibility as a rule is not affected; but in some cases over a limited area of one of the limbs there is some loss of tactile sensibility.

Sight.—Besides the transient diplopia which occurs as an early symptom, primary optic atrophy very frequently occurs, often in one eye before the other, and is attended with loss of sight. In some other cases, but less frequently, the atrophy is preceded by a slight neuritis.

The sphincter ani and vesicæ are usually not affected till late in the disease.

Mental changes, such as undue emotion, laughing and crying without any definite reason, are met with in the early stages, and later on dementia occurs.

Vertigo is described as occurring in many cases (Charcot), associated with vomiting.

Other symptoms which occur late in the disease are atrophy of groups of muscles, bulbar paralysis, and apoplectiform attacks with a quick pulse and a rapid rise of temperature.

Pathology.—The symptoms are due to patches of sclerosis scattered over the central nervous system, and affecting especially the white matter of the hemisphere—the centrum ovale—the cerebellum, pons and medulla, and the spinal cord. The cranial nerves are sometimes involved, but rarely the cortex cerebri. These patches are of a reddish grey colour of the size of from a pin's head to that of a walnut, and on microscopical examination they consist of an overgrowth of the neuroglia with degenerated nerve fibres; the patches gradually enlarge, and three zones can be made out,—at the periphery, where the nerve fibres are separated by the overgrowth of the neuroglia; at the intermediate zone, where the nerve fibres have lost their medullated sheaths, retaining only the axis cylinders, which are often enlarged; and an internal zone, the oldest part of the disease, occupied by thick bundles of connective tissue of the neuroglia, with a few axis cylinders, fat cells, and granulation corpuscles. When the disease spreads to the grey matter, the nerve cells are first affected with yellow degeneration and then they atrophy. The pathological changes seem therefore to begin by an overgrowth of the neuroglia, by which the nerve fibres are secondarily compressed and destroyed. With regard to the symptoms, the inco-ordination is due either to interference by the patches with the proper conduction of the motor impulses along the white fibres (Charcot), or to affection of that part of the brain where a particular movement is co-ordinated (Erb).

Diagnosis has to be made from other forms of tremors. Of these hysterical tremors, those of hereditary ataxia, cerebellar tumours, and general paralysis of the insane are the most important. The diagnosis from hysteria is of the greatest importance, and it is often very difficult and sometimes impossible, but the presence of nystagmus, of ankle clonus, and the complete cessation of the tremors during rest would make the diagnosis of sclerosis fairly certain; from hereditary ataxia, disseminated sclerosis is distinguished by the knee jerk being excessive, by ankle clonus, by the larger movements of the inco-ordination of the arms, and as a rule by their not being increased by closing the eyes; from cerebellar tumour, by the scanning speech, by the absence of headache, vomiting, and optic neuritis, and by the large excursions of the cerebellar reel—*i.e.* a case of disseminated may have an unsteady walk, but the patient does not tumble bodily over to one direction as in cerebellar cases; in general paralysis of the insane without mental exaltation, the pupils are unequal and often do not react to light, further the over-action of the facial muscles, the "smudged" speech, the absence of nystagmus, and the dementia would separate general paralysis from sclerosis.

Prognosis.—The ultimate prognosis is bad; but the disease, instead of progressing gradually worse, often has remissions in which there is marked improvement, though later the symptoms recur until the patient becomes bedridden. If there are no remissions, death usually occurs in from two to three years; but with remissions it may last several years. Three stages of the disease are described: (1) from the commencement of the first symptoms till the patient is unable to leave his bed; (2) when he is obliged to keep his bed, but is able to control his bladder and bowels; (3) when he has lost control of the sphincters, has become quite feeble, and has developed bed sores, ending in death.

Treatment.—As unfortunately there is no drug which directly influences the disease, the best treatment is to keep up the patient's strength with nerve tonics, as liquor arsenicalis, beginning with ♏ij, and gradually increasing the dose. This has seemed in some cases to cause improvement, but as the disease is liable to periods of improvement, it is difficult to say how much is due to the treatment. Strychnine, quinine, and iron may be given, also cod-liver oil and maltine. The patient should be encouraged to go about as long as he can, but to avoid all worrying and depressing influences. Electrical treatment as the constant current, and various forms of baths have been used; but it is doubtful if any real improvement is obtained.

In the second and third stages, when the patient is bedridden, great attention should be paid to prevent bed sores by the use of a water bed; and if there is any retention of urine, a catheter should be passed daily and the bladder washed out with boracic lotion.

HYDROCEPHALUS.

This consists of an accumulation of serous fluid in the ventricles (internal hydrocephalus), or in the subdural space (external hydrocephalus); and the condition may be acute or chronic.

Acute hydrocephalus is usually the result of an acute meningitis.

Chronic hydrocephalus is sometimes external affecting the subdural space, but by far the commoner variety is the internal, where the ventricles are distended with fluid.

Two varieties are described—*congenital*, which may originate *in utero* and obstruct parturition, and of which the cause is unknown; and *acquired*, which is usually due to some pressure on the veins of Galen from a tumour, or from tubercular granulations resulting from an attack of meningitis, or it is due to closure by meningitis, sometimes from congenital syphilis, of the foramen of Magendie, the normal way by which the fluid escapes from the fourth ventricle into the subarachnoid space.

Symptoms are mental weakness, some paralysis or contracture of the limbs of one or both sides, epileptic fits, vomiting, nystagmus, strabismus, and blindness with optic atrophy from pressure of the distended third ventricle on the optic chiasma. The child is often very irritable, and in congenital cases idiotic. The weight of the head is sometimes so great that it cannot be raised up. The knee jerks are usually increased.

The head becomes very much enlarged, and the sutures become separated; the fontanelles are distended; the head becomes rounded, making the face look very small; the roof of the orbits is depressed, making the eyeballs prominent. When the fluid is in the ventricles, the cortex and centrum ovale are compressed against the skull and thinned and the cortical convolutions are flattened. If the foramen of Magendie be patent, the fluid escapes into the subarachnoid space of the spinal cord, which is thus distended.

Diagnosis.—If the fontanelles have not closed, the diagnosis is made by the fact that they are bulged by the pressure of the fluid. If they are closed, the diagnosis has to be made from rickets and from thickening of the cranial bones, by the presence of these conditions elsewhere in the body.

Prognosis is usually bad, and death occurs in severe cases in a few months; but a few cases have recovered.

Treatment consists in keeping up the strength of the child by careful feeding, and syrup of the iodide of iron and cod-liver oil.

Evacuation of the fluid has been performed in several cases, and may be done through the anterior fontanelle by a long trocar passed down through it into the lateral ventricle; or by continuously draining the descending cornu of the lateral ventricle, by trephining over the temporo-sphenoidal convolution and passing horizontally in a trocar and leaving the canula in to drain; or by puncturing the spinal membranes in the lower lumbar region and draining continuously. Compression of the skull by strapping or by an elastic bandage has been employed after tapping. The results are not very encouraging, but if the pressure is extreme, tapping with strict antiseptic precautions may be tried.

CHAPTER XIX.

ORGANIC DISEASES OF THE BRAIN

(continued).

AFFECTIONS OF SPEECH.

APHASIA.

To understand the mechanism of speech we must examine what takes place with a child learning to talk. The child has a vocal apparatus by which different sounds can be imitated, but these sounds must first be heard; it is therefore through the auditory apparatus that sounds of words spoken to the child are heard by it and imitated. Hence speech is at first entirely auditory. Later on the sound, as "pa-pa," is associated with the visual impression of the father, and in time this visual impression will call up and reproduce the name, which the child will then say. Here the visual impression is able to act on the auditory centre and call up the sound of the name of the object seen. Later still the child is taught that certain symbols called letters are used to denote certain sounds, so that the visual impression of the letters "PA-PA" can call up in the auditory centre the sound which expresses the name of a certain individual. Subsequently the child is taught to copy these symbols, and to associate them with certain sounds; this is writing.

As this is such a complicated subject, we will first describe the method by which the degree, to which the patient has lost his speech, is found out, and then the chief different forms of loss of speech.

Bound up with the question there are, besides the power of articulating intelligibly, the power of understanding language which the patient hears, and the names of objects and words which the patient sees, as well as the power of writing. We must therefore ascertain the following points:—

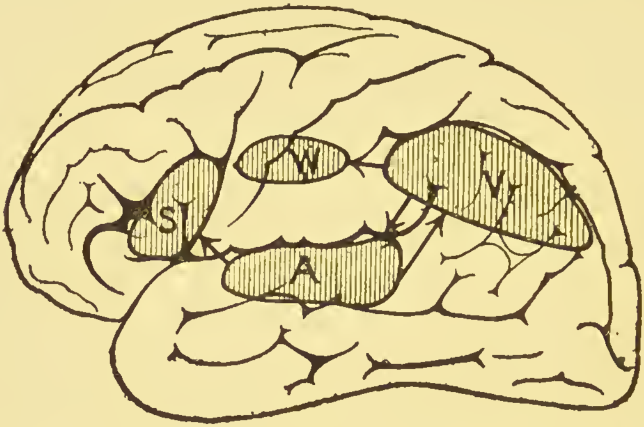

Fig. 27.—Diagram of left cerebral hemisphere, showing the parts of the cortex affected in aphasia. S, the speech centre of Broca; A, the auditory word centre; V, the visual word centre; W, the probable seat of the centre for writing. The arrows denote the commissures between the centres, and the direction in which the nerve impulses pass (after Bastian).

1. Can he spontaneously *speak* intelligible words? . . . . . . (S)
2. Can he understand commands which he *hears*? . . . . . . (A)

3. Can he understand written commands which he *sees*? . . . . (V)
4. Can he *write* spontaneously? . . (W)
5. Can he copy from printed to written characters, *i.e.* what he *sees* and understands? . . . . . . (V...W)
6. Can he write from dictation what he *hears*? . . . . . . (A...V...W)
7. Can he pick out objects and letters of which he *hears* the name? . (A...V)
8. Can he repeat the names of objects or letters *heard*? . . . . (A...S)
9. Can he name objects or words *seen*? Can he read aloud and understand? (V...A...S)

In the above list the capital letters in brackets show the parts of the brain involved in performing each question.

Besides the above, it is well to ascertain if a patient understands the use of an object, even if he does not know the name of it. The above list of questions is for the purpose of finding out which parts of the brain are affected, and it is divided up into three groups of threes: in the first group are the primary centres for speech, word audition, and word vision; then the three forms of writing; and last the power of picking out, repeating, and naming objects and words.

There are four principal parts of the brain which may be involved: (1) the speech centre of Broca, situated in the posterior part of the left third frontal convolution (fig. 27, S); (2) the auditory word centre in the first temporal convolution (A); (3) the visual word centre in the angular convolution (V); (4) the centre for co-ordinating the movements of writing (W), probably in the region for the representation of the hand in the left ascending frontal and parietal convolutions. These centres are connected by fibres, as shown in the diagram. For instance, in writing from dictation, the person hears a word or sentence, which is received by the auditory centre (A); this calls up a mental picture in the visual centre (V), which is carried on to the centre for writing (W), and directs the correct writing of the word or sentence.

In right-handed persons all the above centres are situated in the left hemisphere, and a lesion of the right hemisphere will not produce aphasia, except in left-handed people when it is the right hemisphere and not the left which produces these speech defects. Aphasia is produced by lesions of the cortex only, and not by lesions of the white fibres of the internal capsule; the usual explanation of this is that, in the former case the corpus callosum being affected, communication is cut off between the speech centre in the left hemisphere and the motor centres for the tongue, larynx, etc., in the right cortex, while in the latter case speech can still be carried on through the corpus callosum and the right cortex.

In examining a case of affection of the speech, all the above questions should be asked, and the pen put through those the patient cannot do. In the questions 5-9 the letters S, A, V, W are connected by dots, to denote the fibres joining these centres; for it is possible that the lesion may be in these fibres and not in the centres themselves. This would be shown by the patient being able to fulfil the first four questions, and failing in some of the others. It must be remembered that all degrees of failure are met with, and in one or all the above questions the patient may be defective.

There are, then, four chief centres which are employed in forming one's idea of words: the auditory word centre, by which a word can be recognised from its sound; the visual word centre, by which a word can be comprehended from its appearance in printing or writing. These two are sensory memories, and to make use of them two motor efforts are required—*viz.* the centre for the representation of the movements of the tongue and lips for articulation, and a centre for the co-ordination of the muscles for writing. As shown in the diagram, the speech centre is connected with the auditory centre and the centre for writing with the visual centre. There are also four chief disturbances of speech.

The word "aphasia" was originally given to the simple cases where there was only loss of speech. This, which now goes by the name of—

(1) *Motor aphasia*, or ataxic aphasia, is caused by a lesion of the speech centre (S), or Broca's centre—*i.e.* the posterior part of the third frontal convolution of the left side. On examination it will be found that the patient cannot answer any of the questions in the above list into which S enters; he will not therefore be able to speak intelligible words, he will not be able to repeat names of objects or words, either spoken to him, or shown to him. He will be able to perform commands spoken or written down, he will be able to accomplish the three forms of writing, and to pick

out objects and letters named to him. These cases can usually make some incoherent sounds, showing that the laryngeal apparatus is not at fault; many can say "Yes" and "No," or only expressions as "Ba-ba" to everything, and although when agitated or annoyed they come out with the expression "Oh dear!" or swear, they cannot repeat the expression directly after, showing that they are automatic emotional utterances and not voluntary.

(2) Inability to write, or *agraphia*, has been observed to occur alone, and I have one case under my care, but is more often associated with other forms of aphasia. When the part of the brain (W), which is probably near the representation for the hand, where the movements for writing are co-ordinated, is affected, all forms of writing are lost. The power of writing depends most on the visual word centre primarily, as even in writing with the eyes shut there is a mental picture of the words which directs the hand. In cases where the visual word centre (V) is affected, the patients may be able to write their names or copy printed words as such, but they are quite unable to read or understand what they have written (*i.e.* like a person copying Chinese characters), and as a rule they cannot write from dictation.

Sensory aphasia, or amnesia. Of this there are two chief kinds, depending on whether the auditory or the visual word centre is the more affected.

(3) *Word deafness.* This is the name given to cases where the auditory word centre (A), situated probably in the left first temporo-sphenoidal convolution, is affected. In this case all questions into which (A)

enters cannot be performed. These patients can hear ordinary sounds or music, but they have lost the power of recognising the sound of names of objects, or of letters, and they are unable to understand commands reaching them through the sense of hearing; they cannot write from dictation; they cannot pick out objects or words named to them, and cannot repeat the names of objects or letters when they are mentioned to them: they can usually read correctly and understand short sentences; they can talk fluently, but the words are often mixed up and nonsensical, although they are not aware of this; they can write spontaneously and copy.

(4) In the other form, called *word blindness*, the lesion is in the visual word centre, situated probably in the left supra-marginal or angular convolution, at the posterior end of the Sylvian fissure; the patients cannot understand impressions coming through visual channels, and they will not be able to carry out the questions in the list containing V.

They therefore are not able to understand written commands, to copy printed to written characters, to write from dictation, to pick out objects or letters named to them, to name objects or words seen, or to read aloud. They can, however, talk well, can perform all commands spoken to them, can write spontaneously, can sometimes write from dictation—as it is possible that a person can write without using his visual word centre—and though they can repeat the names of objects or letters heard, they cannot recall them a moment later.

With regard to the connecting fibres, between the auditory and visual centres there are two sets which go in opposite directions; for instance, in inability to write from dictation (question 6, A...V...W) the lesion may be in the fibres going from the auditory to the visual word centre; whereas inability to name objects or letters seen may be (question 9, V...A...S) caused by lesion of the fibres going from the visual to the auditory word centre.

Mechanical Difficulties in Articulation.

These must be carefully distinguished from aphasia, both motor and sensory. Here the patient is usually able to speak and say any word, but the pronunciation is thick and faulty, and is often associated with some paralysis of the lips and tongue and with left hemiplegia; he can always write, and he has no difficulty in executing commands, either spoken or written down. In other cases there has been a double hemiplegia, first on one side and then on the other, and this has been followed by complete loss of speech, even for vowel sounds. Paralysis of both vocal cords and of the tongue and soft palate in bulbar paralysis causes loss of all speech and all phonation except a slight grunt, so that the patient has to write down all he wants to say. In these cases the intelligence is sharpened, and they show no signs of aphasia, besides, it is unusual for aphasics not to be able to say one word or expression, especially under emotion; the wasted tongue, motionless palate, and the inactive vocal cords will make sure the diagnosis of bulbar paralysis.

Prognosis in aphasia differs very much, and it is often quite impossible to give a definite answer. A certain number recover their speech by degrees, and others do not improve at all, even twenty-five years after.

Treatment.—The general treatment is that for hemiplegia. The special treatment is to teach a patient his letters and spelling like a child, and the best means are the letters printed on separate pieces of wood, by which the patient can practise spelling out words. If the right hand is paralysed, he should be encouraged to use the left for writing.

CHAPTER XX.

FUNCTIONAL DISEASES OF THE BRAIN.

EPILEPSY.

THIS is the name given to sudden attacks of loss of consciousness with or without convulsions, and not depending primarily on failure of cardiac action, on diseased blood conditions, as uræmia, or on organic disease of the brain, as tumour. These conditions may produce convulsions; but the term "idiopathic epilepsy" is kept for that form of this disease in which no organic changes have hitherto been found.

Etiology.—*Predisposing causes. Hereditary* tendency to epilepsy or insanity is found in one-third of the cases (Gowers). *Age:* a very large proportion begin under twenty, and most about puberty, after twenty the cases diminish, but they may first occur past middle life.

Exciting causes are mental, as sudden fright, extreme prolonged grief and anxiety; or physical, as blows and falls on the head, which sometimes produce general convulsions; or reflex irritation, as intestinal worms, and rickets associated with teething fits of children. Dyspepsia and constipation also act as causes. Delayed menstruation is often supposed to be a cause, and parents are often told that the fits will pass off when this function is established; this, as also the mystic age of twenty-one, when similar hopes are encouraged, do not really have any effect on the cessation of fits. Masturbation is also probably as much the result as the cause of the fits. In a certain number of cases no cause can be found.

Symptoms.—Epilepsy occurs as slight attacks or petit-mal, and severe attacks or haut-mal. Consciousness is lost, but the period in the attack at which this occurs varies, and consequently if it is not lost at the very beginning of the attack the patient experiences certain sensations of the fit, and these constitute the warning before an attack.

The *slight attacks* or minor epilepsy are characterised by a complete loss of consciousness without any definite convulsions. There may be no warning, or the patient may complain of feeling giddy, of general startings of the whole body, of a feeling of some impending danger, or may feel an epigastric sensation which may pass to the left side of the chest or up to the throat and so to the head, or some of the visual sensations (described below) are experienced; consciousness is almost invariably lost, the face turns slightly pallid or flushes, a moaning noise is made, the eyes are wide open and fixed, the pupils dilated and not reacting to light, and urine may be involuntarily passed. These symptoms may occur without the patient ever falling down, or they may fall down anywhere, hurting themselves. In very slight attacks the patient pauses, looks strange, and then goes on with the conversation; and they call the condition "blanks" or "forgets," etc. These attacks last from a few seconds to half a minute, and after them the patient looks dazed, and then goes on with what he was doing, recovering almost immediately. In other cases the patient passes into an extraordinary condition, which has been termed "masked epilepsy," and in which he performs unconsciously automatic actions, which may vary from simply undressing himself in public to walking or driving about or even going by train for some hours without the slightest recollection afterwards. Sometimes automatic actions of which the patient has no recollection, such as running fast, are the first symptoms of a fit. These cases are naturally of great medico-legal importance, as robbery or other crimes may be committed in this condition.

The *severe fits*, called also "haut-mal," "major epilepsy," may begin with a warning, or in about half the number of cases loss of consciousness may be the first symptom.

The warning may be—(1) Psychical, where some idea comes into the patient's mind, or everything seems strange, or he feels as if in a dream, or has some horrible sensation of impending danger which he cannot describe.

(2) A feeling of vertigo, in which objects appear to revolve, or the patient feels himself turning round.

(3) Motor or sensori-motor, where the patient has tingling, numbness, or pain in a definite part of the body, such as the thumb or big toe, and which runs up the limb. This is followed by "drawing up," or tonic contraction of the part where the tingling began; and in some cases clonic contractions are felt before consciousness is lost. In other patients the tonic

contraction is the first symptom felt, and there is no sensory warning.

(4) Beginning in one of the special senses, especially the visual, and less frequently the auditory, the warning may be of two kinds—(*a*) flashes of light and stars, which may be coloured, before the eyes, or noises in the ears; (*b*) definite pictures or spectres. One patient always saw large buildings, another a farmyard with golden fowls, and a third a group of men in a circle the last of whom hit the patient when he had a fit; or a definite tune as the "Marseillaise" was heard in one case, or a voice calling the patient over his shoulder in another. The latter group are more complicated and more highly evolved, and probably take place in much higher centres than in the case of flashes of light and noises. In rare cases olfactory or gustatory warnings may be experienced.

Patients may experience the warning without the fit being developed and without loss of consciousness.

Character of attack.—In a severe fit the convulsion can be divided into two stages, the tonic and the clonic; but there is no hard-and-fast line between them, and one runs into the other.

Sometimes the aura is described as a stage; but there is no cause for this, as an aura only means that consciousness is not lost at once, so that the patient is conscious of the first part of the fit.

A fit usually begins suddenly, but for some hours previously the patient may have had startings over the whole body. The attack begins sometimes with a cry, often an unearthly scream, and the patient falls down often forwards, sometimes backwards. The tonic spasm then appears to affect the whole body, but if a fit is carefully observed at the very commencement, the spasm is usually seen to involve one side of the body more than the other, so that the face is turned to that side; the eyes are drawn conjugately to the same side, and also the corner of the mouth is retracted on that side; the fingers are pressed against the thumb, and the elbow is flexed; the legs are usually flexed at the hip and knee, but the limbs on the side to which the face turns are usually the more affected, and the spine is sometimes flexed towards that side. The muscles of respiration are fixed in the tonic spasm, and as a result the face becomes cyanotic; the pupils do not react to light.

The tonic spasm gradually passes into the clonic, which consists of a rapid series of contractions of the muscles, which gradually become slower and larger in amplitude, the eyes are rapidly jerked towards the side to which they were turned in the tonic spasm, the tongue is protruded and caught between the jaws closing spasmodically and bitten. Urine and less often fæces are passed involuntarily, and the pupils are dilated and inactive to light. In some cases the pulse is slowed and becomes irregular, but the alteration of the pulse is certainly not the starting-point of the fit, and is a symptom and not a cause.

After the convulsions have ceased, the respiration is laboured, the cheeks being puffed out, and foam stained with blood is blown from the mouth, the face is cyanotic and often covered with sweat, the limbs are lax or sometimes rigid, the eyes slowly turn away and deviate conjugately towards the side opposite to which they turned in the fit, the pupils are inactive to light and oscillate, contracting and dilating, the conjunctival and the superficial reflexes are abolished. The knee jerks are increased or are absent when unconsciousness is profound; but in all cases they soon become increased with ankle clonus, and this is especially marked on the side which was the more convulsed in the fit. It is on recovering from this stage that patients may occasionally pass into a state of mania, or perform the automatic unconscious actions described above. After a fit in which one side is much more convulsed, this side may be temporarily paralysed, and after some right-sided fits aphasia may result for a time.

The *duration* of the fit varies; but the tonic stage lasts as a rule from three to five seconds, and the clonic stage for from thirty to ninety seconds, or in some cases longer. After all convulsion has ceased, the patient remains unconscious for about five minutes, when the conjunctival and other superficial reflexes return, ankle clonus disappears, and the knee jerk resumes its normal condition; he gradually recovers his senses, opens his eyes, and looks about him, and in another five or ten minutes may be able to answer questions and walk. In other cases patients pass into a deep sleep.

The *frequency* of the fits varies very much. Attacks of petit-mal may occur every day, or several in one day; and the severe fits every day, but more frequently once or twice a week or a month. When the severe attacks occur with great frequency several times a day, the patient has not time to recover his senses before the next attack comes on, and he passes into the "status epilepticus"—a very grave condition—in which the temperature rises, and the patient may die if unrelieved.

After the patient has had the first fit, the next one may occur in the course of the next month, or it may not come on for another year, or it may be for several years.

The *time* of day at which fits come on varies, occurring at any time, during the day, or only at night-time, or when the patient is getting up in the morning. The time of the month, with the exception of just preceding the menstrual period, does not have any effect, and the popular belief in the occurrence at the full moon is not borne out by careful investigation.

The *mental condition* between the fits may be perfectly good; in some memory is bad for recent events, while in others the intellect becomes defective down to dementia. In many cases the defective condition seems to be caused by the length of time the fits have occurred, and especially by their frequency and severity; in others, especially children, it is another expression of the defective cerebral development which produces the fits.

Pathology.—Nothing has hitherto been found in the brain to account for idiopathic epilepsy; but it is highly probable that all cases have their seat in the cortex, from the fact that tonic spasm followed by clonic spasm can be produced experimentally only by stimulating the cortex cerebri. The character of the movements of epilepsy is also precisely that obtained by electrical stimulation of the motor cortex. The muscles of the opposite half of the body are put into action except those for turning the head. In a fit affecting the right side the more, the turning of the head, so that the face looks to the right, is done by the *left* sterno-mastoid, and as a right-sided fit is caused by irritation of the *left* cortex, the *left* sterno-mastoid is acted on by the *left* cortex. The movements in epilepsy are those of physiologically grouped muscles, and it is only as an anatomical convenience that the muscle on the left side is used to produce a movement of the head to the right. The cells of the cortex must be regarded as pent-up reservoirs of energy, and in a healthy person the cell is able to keep its energy under proper control; but this control is broken down by the shock of a blow on the head, or the irritation of a depressed fracture, and by the intravenous injection of absinthe, and the person has a fit. In epileptics the cerebral cells are probably congenitally weak, and periodically, or under any exciting cause, their energy cannot be kept under control, and produces a fit. The part of the cortex where the fit begins is shown by the warning, or the first spasm; for instance, a fit beginning with a sensori-motor sensation, *e.g.* tingling followed by tonic and clonic spasms of the thumb, would have its seat in that part of the ascending frontal and parietal convolutions where the movements of the thumb are represented; a "special sense" aura would begin in the part of the cortex where that particular sense is localised, while attacks of petit-mal would take place in the part of the cortex where the highest mental processes occur, probably the præfrontal region.

Diagnosis.—When a fit can be seen, there is not as a rule any difficulty about the diagnosis, but so often an opinion has to be given merely on the description of the friends. The chief characteristic symptoms of a severe attack of epilepsy are the sudden falling down anywhere, *i.e.* into the fire, tongue-biting so as to make it bleed, involuntary passage of urine or fæces, and the non-reaction of the pupil and conjunctival reflexes showing the unconscious condition during the attack.

From *uræmic* fits epilepsy cannot be distinguished, except by the presence of albumen in the urine and other cardiac and ophthalmoscopic appearances of Bright's disease, as in idiopathic epilepsy there is no albuminuria, or only after very frequent fits, and no ophthalmoscopic changes.

The diagnosis of idiopathic epilepsy from epileptiform fits due to a cerebral tumour is occasionally very difficult, if not impossible, when the former begin with a very local warning, and affect only one side of the body, and when the latter are attended with loss of consciousness. The presence of optic neuritis, headache, and vomiting will make the diagnosis of a cerebral tumour almost certain; but in the absence of these symptoms, a tumour must be suspected if the fits are very frequent, are not affected by drugs, and always begin in the same place, and more especially if paralysis in the convulsed limbs persists and is not temporary after the fit.

From *hysteroid* fits it is not difficult to make a diagnosis if the fit is witnessed. In these cases the patient does not fall so suddenly or hurt herself, and she may scream or talk at intervals during the fit (an epileptic screams only at the beginning); there is no definite tonic followed by clonic convulsion, but there is much violent purposive struggling and resistance to being held. These patients do not bite their tongues, but their fingers and the people holding them. The eyelids are tightly closed, and resist opening; the pupils react to light, and the conjunctival reflex is present; there is little or no cyanosis of the face, the excreta are not passed, and on applying strong stimuli as faradisation the patient recovers her senses quite suddenly.

In some cases hysterical fits follow on slight epileptic fits. Here the non-reaction of the pupils at the very onset will help to decide if the first stage is epileptic.

Attacks of petit-mal have to be diagnosed from cardiac syncope. Where there is distinct failure of the heart's action, the patient if unconscious always falls down, there is no involuntary passing of excreta, and there is always some premonitory warning of "faintness," which may be quite absent in petit-mal. Also from auditory vertigo, which it is difficult to diagnose from petit-mal beginning with an auditory aura or with an aura of giddiness; but in auditory vertigo there is never complete loss of consciousness, though the patient may feel dazed, and no involuntary micturition.

Prognosis.—Epilepsy as regards life is not serious if the patient is careful to avoid dangerous places as machinery, water, etc., where he might fall down and be killed; but some cases always turn on their face in bed, and are liable to be suffocated.

As to whether the fits will recur, it is quite as impossible to promise that a person who has once had an attack will never have another fit, as it is that he will never have another headache; but the longer that he has gone without a fit, the more the chance of the fits not recurring. Fits beginning after twenty are more favourable than those beginning before that age. The prognosis is worse when there are a large number of slight fits and when mental changes are present.

Treatment.—With regard to general treatment, patients and their friends should be impressed with the importance of their avoiding dangerous places, where a sudden fit would risk the patient's life; also it is important that some one should sleep in the same room. An outdoor life with moderate exercise is most beneficial; and though excessive mental work and worry should be avoided, it is a great mistake to let epileptics be idle and learn nothing, and they should be educated within their powers. Marriage does not seem to have any effect, but it certainly does not diminish the fits, and the question of the probability of the children having epilepsy or some other nervous disease should be pointed out.

No particular food, provided that it does not cause indigestion, is required; but some epileptics will eat to excess, and especially of meat, and this must be prevented. Stimulants in moderation do not have any effect either way. The bowels must be particularly attended to, as constipation may easily bring on a fit.

In a fit the patient should if possible be caught as he falls, and laid down flat on the ground with a pillow under the head; all tight clothing about the neck and chest should be loosened, and a spoon wrapped round with a towel, or a piece of india-rubber inserted between the teeth to prevent the tongue being bitten; the patient's limbs need not be held, as the movements are not sufficient to hurt them; they should be allowed to sleep after a fit.

With regard to medicine, the bromides are undoubtedly the most reliable. It has been recommended to give three salts of bromide at the same time; but there does not seem to be any special advantage in this. It is best to give one salt of bromide, the potassium or ammonium salt, in just sufficient quantity to control the fits—*viz.* from gr. x to xx three times a day, or gr. xxx night and morning. If anything, the ammonium salt has perhaps less depressing action than the potassium salt; but it is good to vary the salt used. Generally, if a drachm and a half of bromide in the twenty-four hours will not modify the fits, it is doubtful if a larger dose will be of use, but still it may be tried. The time of day of giving the dose is important. If the fits are only at night, a full dose of gr. xl or ʒj should be given at bedtime only; if they occur in the morning on getting up, gr. xxx or xl should be taken at that time, and the patient stay in bed for an hour after. If the fits occur any time, gr. xx three times a day, or gr. xxx twice a day. Patients taking bromides are liable to become "bromised"—*i.e.* to pass into a demented condition, with dribbling of saliva from the mouth, slow pulse, and cold extremities—when the bromide must be stopped at once; or liable to have bromide acne, which is usually prevented by taking ♏ij to ♏v of liq. arsenicalis with each dose.

Belladonna in 5-minim doses of the tincture added to each dose of bromide is useful, and also lactate of zinc gr. ij to gr. viij after food, especially with attacks of petit-mal.

Borax gr. xv to xxx three times a day after food has been found to answer when bromide has failed, and also nitro-glycerine in doses of $\frac{1}{200}$ grain up to gr. $\frac{1}{75}$, or $\frac{1}{2}$ minim to $1\frac{1}{2}$ minims of the tinct. trinitrini three times a day after food (Gowers).

Digitalis in 5 to 10 minims of the tincture when the circulation is weak, and tinct. cannabis Indicæ in 10 to 15 minims, in conjunction with bromides, are useful when bromides fail alone.

In using the above drugs, it is of no use taking them for a month or two. The patient should be impressed that on no account should he leave off taking the medicine, not only while he has the fits, but for a year at least after the last fit; and then it should be left off gradually, taking half of a dose for three months, and quarter of a dose in the next three months, and leaving it off completely by degrees, as even after cessation for a year the fits may return on discontinuing too suddenly the bromide.

It is often impossible to prevent a fit, but if the patient has warnings and knows for half or quarter of an hour before a fit comes on, a full dose of bromide may be taken in the hopes that it will be absorbed in time to stop the fit. In more rapidly occurring fits nitrite of amyl carried in the pocket in glass capsules and inhaled has stopped fits in some instances. Fits beginning with a warning in the hand have been prevented by suddenly tightening a ligature round the upper arm, and so checking the sensation, which does not pass the ligature. A circular blister to the wrist has the same effect, also any counter-irritation, as a blister or seton to the neck, will often diminish the fits.

The condition known as status epilepticus, in which the patient passes from one fit to another without recovering consciousness, is best treated by an enema of bromide and chloral gr. xv to gr. xx of each every three hours, or by the inhalation of chloroform, which often gives only temporary relief.

Surgical treatment should be employed in cases where the fits are very frequent and are not kept under by the bromides and begin with a definitely localised warning, as tingling or spasm of the thumb; the skull should be trephined over the exact point in the cortex where the fit begins, and if nothing abnormal is found, it is justifiable in severe cases to remove that piece of the cortex in which the fits begin; this part is found by stimulation by a weak faradic current of the cortex and finding the exact point which gives the movement with which the fit begins, and removing that part, a method which has been employed by Horsley.

Other forms of convulsions are very like epilepsy in their symptoms, but they arise from different causes Of these puerperal and uræmic convulsions are due most probably to the presence of waste products in the blood, which the diseased kidneys are unable to excrete and these waste products probably act as an irritant to the cortex cerebri, producing fits characterised by tonic and clonic spasms. The fits often affect first one side of the body only, and then involve the other side, and in the case of uræmic convulsions the clonic stage may last for hours. For the treatment of these cases, reference must be made to works on obstetrics and on Bright's disease.

INFANTILE CONVULSIONS.

These occur most frequently from the age of a few months to the end of the second year; but in some cases, they persist, or they recur at seven years old, and may pass on to active epilepsy.

Causes.—Teething has been put down as a certain cause, but as many children have painful dentition without having fits, there must be some predisposition, perhaps hereditary, of the nervous system to instability. Rickets, as shown by Sir W. Jenner, is a much more potent cause. Other exciting causes are direct irritation by food, lumbrical worms, etc., of the intestines. Convulsions in children may take the place of a rigor in adults at the onset of one of the acute specific fevers.

Characters are very like those of epilepsy, except that they are less severe, and are often more local, involving first one side or part of the body only, and then the other side. The eyes become fixed, tonic spasm occurs in the affected part, followed by clonic spasm, the face becomes cyanotic owing to the respiration ceasing, and involuntary micturition and tongue-biting are unusual. The tonic stage often lasts longer than in epilepsy—a minute in place of five or ten seconds. Attacks of laryngismus stridulus are considered by many authorities to be a tonic spasm of the glottis similar to other infantile convulsions, the spasm lasting a few seconds, causing marked cyanosis, and attended by tonic spasm elsewhere, as carpo-pedal contractions, and followed by a loud inspiration when the cords are relaxed.

Treatment consists in removing the cause, as worms and improper food, and improving the general condition by careful feeding with cod-liver oil and the syrup of phosphate or iodide of iron ; and if the fits do not cease, administering bromide of potassium or ammonium, gr. ij to v three times a day to infants, and gr. v to x to older children. Lancing the gums has always been a favourite remedy, and though theoretically it may not do good, many observers testify to its efficacy, and it cannot do any harm.

CHAPTER XXI.

FUNCTIONAL DISEASES (*continued*).

VERTIGO.

VERTIGO, which is also popularly called "giddiness" or "dizziness," is used to describe a sense of movement experienced by the patient, which may be real, and it may be described by the individual (1) as if the room and objects in it moved in a certain direction; (2) as if the patient were moving in a certain direction, either turning round or moving up and down; (3) as if the patient and also objects around him moved, either in the same direction or in opposite directions.

Vertigo is caused by peripheral or central disease. In the former it is produced by a disturbance of the equilibrium of the body or the power of balancing the body in the erect posture; this equilibrium is brought about by impressions received by the brain from the skin, joints and muscles (muscular sense), and by impressions received by the brain from the ocular and auditory organs, including in the latter the semicircular canals. If there is a discrepancy between the impressions brought by these organs, there is a disturbance of equilibrium, and vertigo ensues.

There is thus a vertigo due to loss of muscular sense, as in some cases of tabes; an ocular vertigo, due to diplopia; and an auditory vertigo, due to disease of the semicircular canals, of which there are three in number on either side, and they correspond in direction to the three dimensions of space.

In central disease vertigo occurs as an epileptic warning, and also in some cases of cerebral or cerebellar tumour. Gastric vertigo has often been described as due to dyspepsia; in most cases there is a feeling of faintness and sickness, but no apparent movement of objects, and in the cases where this latter has been observed, it was probably due to an associated affection of the semicircular canals.

Of the above forms of vertigo, the most important is aural vertigo.

AURAL VERTIGO,

Called also auditory vertigo or Menière's disease.

This is caused by a disease of the internal ear, especially of the semicircular canals, and it is characterised by sudden attacks of giddiness, making the patient fall down, and is associated with deafness and noises in the ears.

Etiology.—It is a disease of middle or advanced life, seldom occurring before thirty, and more often after forty, and it affects men more than women.

Causes.—Gout and syphilis have been considered as predisposing to the disease in some cases; in others it seems to be a senile change. Exposure to cold and wet appears to be an exciting cause sometimes.

Symptoms.—The onset of the disease is often quite sudden and unexpected. The patient "feels giddy," and has vertigo, and feels himself falling towards, or turning round to, one side, while objects also may appear to move; he falls down, and feels extreme nausea; he turns pale in the face, and breaks out in a sweat, and at the end of the attack may actually vomit. He may just before the attack have had noises in the ears; but this point has frequently to be ascertained by a direct leading question, as often patients do not associate it with their attack.

Except in very severe cases there is not any loss of consciousness, but the patient feels very dazed and does not know where he is, there are also no convulsions, no tongue-biting, and no involuntary passing of water. In slight attacks the patient feels giddy and staggers, but does not fall down, and after a minute or two recovers; in the severe attacks the vertigo may last for several hours. The attack may be brought on by any violent shaking of the head, by sneezing, or blowing the nose; and the frequency of the attacks may be every week, or with several months' interval between them. There are two symptoms which persist between the attacks—*viz.* noises in the ears and deafness. As one ear is usually attacked before the other, these symptoms are often one-sided.

The *noises* in the ears (tinnitus aurium) are present always and may be intensified just before an attack, or they occur only before an attack. They are described as a continuous hissing, like an engine blowing off steam, or like a whistle, or they are less often intermittent like machinery, and may be synchronous with the pulse.

The *deafness* is usually on the same side as the tinnitus, and is often not complained of by the patient. The essential point is that, owing to the fact that the disease involves the internal ear and presumably the terminations of the auditory nerve in the cochlea, hearing for perosseal conduction through the bones of the skull is lost or diminished, as well as that for aerial impressions ; consequently the distance that the patient can hear a watch from the affected ear is less than on the normal side, and a tuning-fork applied to the teeth is heard less in the affected ear, also a tuning-fork placed on the mastoid process of the affected side can still be heard on that of a healthy person after it has ceased to be heard by the patient.

With regard to the attacks, the patient usually falls towards the side on which there is deafness and tinnitus, and in the vertigo the patient feels himself turning that way ; objects may appear to the patient to go the same way, or more often in the opposite way, but on this point it is often difficult to get a reliable answer.

The attacks may last for many years ; but when the patient is completely deaf, owing to the internal ear being destroyed, the attacks frequently cease.

Pathology.—Experiments on animals have shown that lesions of the semicircular canals produce movements of rotation and instability ; and acute lesions, as inflammation and hæmorrhage into the labyrinth, were found in the cases first described by Menière. It seems therefore highly probable that in the ordinary cases of auditory vertigo there is a gradual degeneration of the membranous labyrinth affecting the cochlea as well as the semicircular canals, and that this may be due to chronic inflammation from cold, gout, or be simply senile.

Diagnosis.—When the attacks are associated with tinnitus and perosseal deafness, there is not usually any difficulty of diagnosis, except in the case of epilepsy beginning with an auditory aura. In the case of auditory vertigo there is often, though not always, persistent tinnitus between the attacks; consciousness is rarely, if ever, lost, and there is no involuntary micturition; perosseal conduction is lost, but it is conceivable that this might also be the case in a tumour in the auditory cortical centre. In epilepsy the auditory warning is not persistent, and only occurs just before an attack, and is not experienced after it. As auditory vertigo is primarily caused by a lesion in the semicircular canals, it is possible that the disease might be present without any tinnitus or loss of hearing.

Prognosis as regards life is not serious; but as regards recovery the prognosis is uncertain, for though some cases are not improved and get quite deaf, others are relieved and sometimes completely.

Treatment.—Any cause as gout, syphilis, or cold must be treated by colchicum with citrate or carbonate of lithia, iodide of potassium, or salicylate of soda; counter-irritants behind the ear are also beneficial. The medicines which do most good are bromide of potassium or ammonium in gr. xx doses three times a day with tinct. belladonna ♏v, and quinine given in doses of gr. v ter die, but larger doses of the last drug have been recommended by Charcot. Electricity in the form of the constant current is useful in some cases of tinnitus. The patient should lie down with the sound ear on a flat electrode connected with the negative pole, and the ear to be treated should be filled with warm water, and an olive-shaped electrode connected with the positive pole should be inserted into the external meatus; a current of from two to four milliamperes, as shown by a galvanometer, should be applied very gradually, and kept on for ten minutes, using a dial collector which does not break the current.

CHAPTER XXII.

FUNCTIONAL DISEASES (*continued*).

NEURALGIA.

The term "neuralgia" has been used in a vague way to denote any painful affection of a nerve; but here the word will be restricted to those cases in which presumably there is no organic disease or structural change, as inflammation of the nerve.

Etiology.—*Age.* The disease is one of adult life, rarely beginning till after twenty, and occurring then any age, but less common in the old.

Sex does not make much difference, taking all classes of neuralgia.

Causes.—*Hereditary.* It is eminently hereditary; according to Anstie, occurring in successive generations. These neuralgic families also have a tendency to severer neuroses, insanity, paralysis, and epilepsy. The patient shows the hereditary taint by being nervous, excitable, and by a tendency to alcoholic excess. Rheumatism and gout are also to be considered as predisposing causes.

Exciting causes.—The patient is nearly always in a weak state of health at the time of the attack. This may be caused by mental worries or overwork, or by a severe shock, or by physical conditions as the puerperal state, overlactation, anæmia, exhausting diseases, malaria, or by alcoholic or lead poisoning. In other cases catching cold or a carious tooth may be the starting-point. Mechanical pressure by a tumour on a nerve may give rise to neuralgia.

Symptoms.—Pain occurring unilaterally and in paroxysms along the course of some known peripheral nerve is the chief symptom. The pains are characterised as shooting, darting, throbbing, and in intensity as burning, boring, cutting, gnawing. They occur in paroxysms which intermit, and between the attacks the patient is quite well and free from pain in the early stages, but usually there is distinct tenderness to pressure on the affected nerve. These points of tenderness are very characteristic, and are especially marked where the nerve leaves a foramen or pierces the fascia. During an attack touching them is unbearable, and between the attacks pressure may bring on a paroxysm. The skin supplied by the nerve is often tender to the touch.

The duration of an attack varies from one single pain to a series of darting pains, which make up a paroxysm ; and this may last several hours, and then intermit.

Other symptoms are occasional numbness in the part, but this not common, and sometimes trophic changes as the colour of the hair turning white (Anstie) ; but herpes is rare in true neuralgia (Gowers), though it follows neuritis.

The different forms of neuralgia may be classified, according to their position, into trigeminal neuralgia, affecting one or more branches of the fifth cranial nerve, cervico-occipital, cervico-brachial, intercostal, lumbo-abdominal, crural; and according to their character or cause into epileptiform, hysterical, traumatic, gouty, diabetic, syphilitic, and malarial.

It will not be necessary to describe all of these. The most important is the trigeminal, which is apt to occur in epileptiform paroxysms. The word "epileptiform" is not a good term, as the disease has nothing to do with epilepsy, except in the resemblance of the suddenness of its attack. The pain comes on very suddenly and very acutely, making the patient call out, and lasting a few seconds or minutes, and then passing off again; probably the pain, with the exception of renal or biliary colic, is more agonising than that in any other part of the body. A paroxysm is brought on by exposure to cold or by any movement, especially that of mastication or talking, and becomes so severe that patients are afraid to eat. Usually one or two of the branches of the fifth nerve are affected, and on one side only, the left perhaps more often than the right.

When the first division is involved, the painful spots are found at the supra-orbital notch about the middle of the supra-orbital ridge, where the supra-orbital nerve leaves the orbit to supply the forehead; also over the supra-trochlear nerve at the inner end of the ridge, and at the junction of the nasal bone with its cartilage where the nasal nerve emerges between them. When the second division is affected, the most painful spots are over the infra-orbital foramen where the nerve issues, over the temporo-malar branch on the malar bone, and along the gums of the upper jaw where the anterior or posterior dental branches course. In the case of the third or inferior maxillary division, the painful spots are just in front of the ear above the zygoma over the auriculo-temporal branch, and over the inferior dental foramen where the nerve comes out to end in the skin.

With regard to the other forms in cervico-occipital neuralgia, the greatest tenderness is over the great occipital nerve between the mastoid process and the occipital protuberance, and also in the posterior triangle of the neck; in the cervico-brachial form there is tenderness over the brachial plexus itself in the posterior triangle and axilla, over the circumflex nerve, over the musculo-spiral nerve just above the external condyle of the humerus, over the ulnar nerve behind the internal condyle, and also in front of the annular ligament at the wrist, and over the radial nerve at the lower and external aspect of the forearm; in intercostal neuralgia the painful points are near the intervertebral foramen, midway in the intercostal space and near the sternum. The sacral and especially the coccygeal nerves are liable to neuralgia. Pains in the nerves of the legs are so frequently due to a neuritis, and not simply to neuralgia, that it is very difficult to say when only the latter exists.

Diagnosis.—The chief symptoms are a unilateral pain occurring along the course of known peripheral nerves, intermittent in character, and often variable in position, and associated with marked tenderness in these nerves between the attacks of pain.

True neuralgia, especially of the head, has to be diagnosed from other forms of pain, and especially from neurasthenic and hysterical pains, and on the other hand from a neuritis or pachymeningitis.

In the former case the pain is more diffuse and like a pressure, and does not follow the course of definite nerves, which are not tender, and the patient complains of curious sensations, as of the head opening and shutting, etc.

In the case of a neuritis, besides the persistency of the pain, there is some evidence of inflammation of the nerve, as anæsthesia, there are trophic disturbances, as wasting, or herpes of the skin, electrical changes in the reaction of the muscles, followed by their wasting, and also often some cause for the neuritis, as a tumour, or aneurysm, or caries of the spine. The pains of pachymeningitis are nearly always bilateral, and are attended with marked wasting of muscles and some anæsthesia. In all cases of pain a careful examination should be made, as otherwise severe organic disease may be overlooked. In neuralgic pains in the legs locomotor ataxia should always be looked for and excluded, and it should be remembered that in unusual cases the lightning pains may affect the head.

Prognosis is very uncertain. It becomes less curable in each successive decade of life (Anstie), and the prognosis is bad for permanent recovery when there is a family history of neuralgia or other neuroses. Cases are more favourable which are due to malaria or to a general weak state of health. Occupation involving exposure to wet or cold will diminish the chances of recovery.

Pathology.—In neuralgia, as opposed to neuritis, there are no organic changes found in the nerves affected. From the fact that division of a nerve often does not stop the pain, it seems highly probable that the morbid condition is not in the nerve trunk, but in the nucleus from which the sensory nerve springs in the pons, medulla, or spinal cord; but what that condition is it is not possible to say. It may be one of instability and hyper-excitability due to malnutrition, so that the sensory cells discharge themselves too readily of their energy, and this is expressed as pain, which is referred to the part of the body from which the nerves come.

Treatment.—This consists in first removing any cause which may be considered to be producing or keeping up the pain, such as a tumour pressing on a nerve, or removing carious teeth in which there is no doubt that the pain originates. This proviso is very important, as one meets with cases in which nearly all the teeth have been extracted and without any benefit, as the teeth were not the cause of the neuralgia, but the pain in them was merely one of the symptoms.

The next most important matter is to treat the general health, and it will very often be found that these patients are ill-nourished, and in many cases this seems due to a want of fatty matters, which these patients frequently object to; the food should contain fatty substances, as cod-liver oil, butter, and cream, or, if these cannot be tolerated, olive oil, which however is not so good.

Other tonics are required, and especially quinine in large doses of gr. v to xx three times a day, and given just before the time that the pain is expected. It acts especially well in cases due to malaria.

Arsenic is useful in ♏iij of the liquor, and in the malarial form Anstie recommends as much as ♏x to xxx three times a day, but owing to the possibility of producing peripheral neuritis doses above ♏x are not advisable, or gr. $\frac{1}{16}$ of arseniate of soda in pill with gr. v extract of hop after food. Valerianate of zinc in 1 to 3 grains has been efficacious in some cases. If there is marked anæmia, preparations of iron as Blaud's pills are indicated, and in gouty cases ♏xv to xxx of the vinum colchici is required. In rheumatic cases iodide of potassium in 5 to 15-grain doses or salicylate of soda diminishes the pain. When the pain is very severe, hypodermic injections of morphia gr. $\frac{1}{6}$ or cocaine hydrochlorate gr. $\frac{1}{10}$ are required, but the patient should never be allowed to use the syringe himself for fear of engendering the habit. Belladonna gr. $\frac{1}{6}$ to $\frac{1}{2}$ of the extract, or atropine hypodermically from gr. $\frac{1}{150}$ to $\frac{1}{30}$ relieves some cases, and extract of cannabis Indica gr. $\frac{1}{4}$ to $\frac{1}{2}$ in a pill, tincture of aconite in 5-minim doses three or four times a day, or aconitine gr. $\frac{1}{150}$ given hypodermically are useful in others. Chloride of ammonium gr. 10 to 20 in intercostal neuralgia and sulphuric ether in visceral neuralgias are recommended (Anstie).

With regard to alcohol, although it often relieves the pain, it should be prescribed with great care in definite quantities, and only at meal-times. Its place can be taken by nitro-glycerine in tabellæ, containing gr. $\frac{1}{200}$ to $\frac{1}{50}$ three times a day (Gowers).

Other remedies which are useful in trigeminal neuralgia are butyl chloral hydrate gr. v to xx three times a day, and tincture of gelsemium ♏x to xx repeated every hour up to three doses, or the alkaloid gelsemine gr. $\frac{1}{60}$ to $\frac{1}{30}$ used hypodermically, also antipyrin gr. v to xx, antifebrin gr. iij to x, phenacetin gr. v to xv, and exalgin gr. iij in pill.

External remedies as counter-irritants in the form of a succession of small blisters over the painful spot, or a mustard leaf, or in some cases the actual cautery, or linimentum chloroformi, relieve the pain.

As sedatives, ung. aconiti may be rubbed in ; oleatum atropinæ smeared over the skin ; menthol gr. xxx to ℥j of lanolin rubbed in ; chloral and camphor may be rubbed together in equal parts, or chloral and menthol, and to these may be added about 2 per cent. of an alkaloid as atropine.

Electricity relieves the pain in some cases. The constant current should always be used, the positive pole being put over the painful spot, and the negative over a point as much as possible in the line of the nerve. In the case of trigeminal neuralgia the negative pole should be at the nape of the neck, the electrodes should be not less than one and a half inches in diameter, and it is absolutely essential that there should be no break in the current. The dial collector of the battery should be arranged that the current can be increased without interruption. The electrodes well wetted with hot water should be first applied without any current, which should then be gradually turned on, and at the end of the setting turned off before removing the electrodes, which should be kept firmly applied while the current is passing. The strength of the current should be measured by a galvanometer and not exceed five milliamperes, which should produce a gentle burning feeling without pain, and be applied for five minutes every day.

Surgical treatment has been employed in severe cases of trigeminal neuralgia which have resisted medical treatment, and especially by Rose, and Horsley. Stretching of a nerve has given relief for from six weeks to two months, but after this period the pain often recurs. Division of a nerve, with removal of a piece of it, has had more permanent results. Rose recommends removal of the Gasserian ganglion; and Horsley, section of the nerve between the ganglion and the pons. Any operation which may give some months' relief, if not permanent cure, to such intense pain, is to be recommended.

MIGRAINE (SICK HEADACHE).

This is an affection characterised by attacks of headache, usually one-sided, often preceded by various optical or other sensations, and followed by vomiting.

Causes.—*Hereditary.* A history of other members of the family having migraine can often be found, or of some allied neurosis, especially epilepsy, and the two diseases sometimes coexist in the same patient; gout in the family is considered to be a predisposing cause.

Age and sex.—Most cases begin between ten and thirty years old, fewer before and after these ages; the sexes are affected about equally—perhaps females are rather more liable. Exciting causes are overwork and worry, prolonged using the eyes in overmicroscopical work, or dyspepsia.

Symptoms.—These may be divided into the stage of sensory symptoms, that of headache, and that of nausea and vomiting. The attacks come on in paroxysms at varying intervals—in severe cases as often as once in every fortnight. The time of the onset is often in the morning, though the patient may have complained of a dull, heavy feeling the day before.

The attack nearly always begins with some *sensory* disturbance, and most commonly this is visual. There may be only dimness of sight, which may be general or hemiopic, affecting the right or the left half of the field in each eye; or the patient experiences a cloud near the centre of the visual field, and this spreads towards the right or left of each field until the whole half-field is blotted out.

In place of these negative symptoms, positive effects are present, sometimes as flashes of light, coloured lights in the centre or to one side of the visual field, or "fortification spectra," when the patient sees zigzag outlines which are often coloured.

In other cases pins and needles followed by numbness is felt in the hand on the same side as that on which vision is affected. This feeling may go up the arm to the shoulder, and is followed occasionally by slight motor weakness. In other cases slight tingling of the cheek, lips, or tongue on one side is felt; and if on the right side, aphasia, motor or sensory, may occur. The leg is rarely affected. These symptoms may be accompanied by extreme paleness of the face with cold extremities.

Headache may come on almost with the sensory symptoms; but as a rule it is delayed till after they have lasted about half an hour. The pain begins over one eye or near the parietal eminence, or over the occipital region of one side, and spreads from one spot over the whole of one side of the head, and sometimes down the neck and arm. It is often very acute, and is increased by movement or by loud noises or strong light. It is said to occur on the side of the head opposite to that of the sensory symptoms. The headache lasts from several hours to a whole day. The pallor of the face may persist, or it gives way to flushing as the headache comes on.

Nausea is not experienced as a rule until two or three hours after the onset of the headache, and usually when the latter is most intense. This is followed by acute vomiting, which does not, however, relieve the headache, and which is quite irrespective of taking food. Sometimes profuse sweating terminates a case. The attack begins as a rule gradually, and passes off in the same way. It often ends in sleep.

In the attack, although there may be slight mental confusion or restlessness, there is never actual loss of consciousness; occasionally giddiness or a "wavy" feeling is experienced as a first symptom. The temperature is not as a rule raised, except in children. In rare cases affection of hearing, with noises in one ear, have been the first symptoms in place of visual sensations.

The duration of an attack, which is allowed to run its course, is from twelve to twenty-four hours, or longer. The frequency of the attacks varies very much; some occur at regular periods, once a week, or once a month—especially at the menstrual time—in other cases less frequently.

Migraine and epilepsy are especially related, and one meets with patients who have both diseases, which may each begin with the same sensory symptoms ; but one patient I have seen in whom no association could be made out between the premonitory symptoms of the two forms of attack. Sometimes epilepsy comes on in a patient suffering from migraine ; in other cases the attacks alternate.

Pathology.—Like epilepsy, no gross changes have been discovered after death to account for the disease. Two theories have been brought forward to explain the symptoms. In the first of these, which has been proposed by Du Bois Reymond, and modified by Latham, the vaso-motor nerves of the sympathetic system are supposed first to cause a contraction of the vessels producing anæmia of the brain and pallor of the face, and next dilation of the vessels giving rise to the headache and flushing of the face. The sympathetic is considered to act without the inhibitory control of the cerebro-spinal system, which is enfeebled.

The other theory, advocated by Liveing, is more likely to be correct—*i.e.* that the symptoms are produced by some alteration in the cells of the cerebral cortex. Probably, as in epilepsy, these cells of the sensory part of the cortex—especially in the occipital region, the centre for sight—are unstable either from overwork, etc., or from hereditary causes, and from some irritation, either from errors of diet or from external causes, as prolonged use of the eyes, the cells are either exhausted, producing hemianopia, or over-active, causing flashes of light and colours.

The explanation is not completely satisfactory, and does not account for the headache and nausea, except as symptoms of a cerebral disturbance. But as in the case of epilepsy, it is necessary to first determine the position of the disturbance, leaving the cause to subsequent investigation. Dr. Liveing would thus ascribe the paroxysms to cerebral storms or explosions, the immediate antecedent being a condition of unstable equilibrium in the parts of the nervous system more immediately concerned.

Diagnosis.—If any attack can be seen, and the symptoms are well marked, the differentiation is not difficult; the sensory symptoms, unilateral headache, followed by vomiting, separate it from other headaches.

It cannot be too strongly urged to ophthalmoscope all cases of migraine. One case, which came complaining of attacks of headache, with dimness of sight and vomiting, very like those of migraine, I found to have optic neuritis, and it proved to be one of cerebellar tumour; but in these cases it is rare to have such long intervals of absence of headache, and the vomiting is more frequent, and occurs without the sequence of symptoms observed in an attack of migraine.

From the aura of an epileptic fit, which is visual in character and is not followed by loss of consciousness or convulsions, it may be impossible to diagnose. In favour of migraine is the longer duration of the visual symptoms, and the fact that they are followed by headache and vomiting. In epilepsy the two latter are not the essentials of an attack, and sooner or later a regular fit will probably take place.

Prognosis.—Cases differ so much that an absolute opinion is impossible, but the prognosis is worse in very frequent attacks which have lasted some time, and where no cause can be made out.

Treatment.—This resolves itself into the management of the condition between the attacks, and into that of an attack itself.

In the interval between the attacks any cause which might produce migraine should be remedied, as errors of diet, dyspepsia, keeping late hours, excessive smoking, or prolonged using of the eyes, as in microscopical work. Errors of refraction, as myopia, hypermetropia, or astigmatism, should be attended to and corrected. With regard to alcohol patients differ, but in all cases anything beyond a very moderate quantity of whisky —which is better than malt liquors—is to be avoided, while other patients are best without any. The general health should be maintained by improving the hygienic conditions when necessary, and regular outdoor exercise should be recommended. Drinking plain water and simple diet seem to have cured some cases, and others have been benefited by a sea voyage.

With regard to drugs, the bromides may be given in 20-grain doses three times a day combined with tinct. belladonnæ or tinct. hyoscyami. Gowers strongly recommends nitro-glycerine given as the liquor trinitrini —a 1 per cent. solution in alcohol—half a minim to begin with, combined with tinct. nucis vomicæ, tinct. gelsemii, and phosphoric acid, or with citrate of lithia and acid syrup. limonis, but it should not be given during an attack. As tonics iron, strychnine, and arsenic are useful, while iodide of potassium in 5-grain doses and valerianate of zinc have been recommended as specifics (Liveing).

When an attack is coming on, the patient should lie down at once in a darkened room kept as quiet as possible, and with warm blankets and hot bottles to the feet if they are cold, and also should take some whisky or brandy. The drugs which have the most effect are antipyrin in 5-grain doses every half-hour up to 20 grains, or phenacetin 5 to 10 grains and antifebrin 3 to 5 grains, which will often check an attack after two doses; but unfortunately they have no effect on some cases, or very soon lose their power. A drachm dose of one of the bromides, with 15 minims of tinct. cannabis Indicæ, and repeated in three hours' time, is useful; also citrate of caffeine in 3 to 5-grain doses, or a strong cup of black coffee or tea.

As local treatment a mustard leaf to the neck often gives more relief than sedative liniments, probably because the pain is not local, but the expression of a central disease. With regard to electricity, a weak galvanic current passed through the head with the positive pole over the painful side of the head has been said to relieve pain; and also statical electricity has been recommended—this is applied by charging the patient when sitting on an insulated stool from a frictional machine, and producing what is known as the electric brush by approaching his head with a pointed brass conductor connected to earth; but as a rule in severe cases patients much prefer to be left absolutely quiet.

CHAPTER XXIII.

FUNCTIONAL DISEASES (*continued*).

HERE are included diseases of the nervous system, presenting symptoms of tremor, and the pathology of which is undetermined.

CHOREA.

The term "chorea" has been used for various affections, and the full term of "chorea Sancti Viti" was originally given to an epidemic of rhythmical hysterical movements, for which also the name of chorea major has been used. The word "choreiform" has also been employed for various forms of habit spasm, and also for movements coming on after infantile hemiplegia. Here the word "chorea" will be used to express chorea minor, or Sydenham's chorea, a disease occurring in children with irregular involuntary movements of one or both sides of the body, and associated with rheumatic changes in the joints and heart, and attended occasionally by some mental disturbance. A form of adult chorea exists, which is hereditary, begins in middle life, and persists; it is sometimes called Huntingdon's chorea.

Causes.—*Hereditary.* In many cases there is a history of chorea in other members of the family, or of epilepsy or insanity, and in nearly one-half the cases is there a history of rheumatism.

Age.—It is distinctly a disease of childhood or youth, occurring rarely before four years, and being most frequent between five and fifteen years.

Sex.—Girls are affected in the proportion of nearly three to one of boys.

Race.—The disease is very rare in coloured races (Weir Mitchell), the proportion being five negro patients to a hundred and seventy-five of whites (Osler).

The season at which chorea is most prevalent has been much studied in America, and it appears to be most in March, and least in November (Morris, Lewis), and to correspond to the prevalence of rheumatism.

Mental influences, as worry or overwork, are looked upon as exciting causes, and also fright, the movements coming on immediately or two or three days after the cause. Imitation from watching another patient does not as a rule produce true chorea, but a hysterical form. Injuries have been considered to produce the disease, but it is difficult to exclude mental influences in these cases. Reflex irritation, as worms, is also a doubtful cause.

Other conditions which are associated with, if not causes of, chorea are acute rheumatism, organic heart disease with or without a history of rheumatism, and pregnancy. Acute rheumatism occurs previously to chorea in about 20 per cent. of the cases; and with regard to cardiac disease, probably in one-half of the cases the heart disease precedes the chorea. Cardiac disease is present in three-quarters of the cases in which there is a history of rheumatic fever, while in a third there is no history (Gowers).

With regard to pregnancy, chorea occurs most frequently in primipara, and about the third month.

Symptoms.—In the earliest stages the child becomes fidgety and irritable, and is often punished for not keeping still. Then there is a difficulty of holding objects, which are suddenly allowed to drop out of the hand, followed by jerking movements of the limbs. Three forms of the disease are described—the slight form, the severe form, and the maniacal form. In the first named the patient is able to walk about; in the severe form the patient is confined to bed, being unable to stand or to feed herself, and the movements may be so violent as to throw the patient out of bed.

In a typical case the hands are the first to be affected, then the face, and later on the legs. In many cases one side is affected before the other, and this is usually the right side. The amount of movement varies very much; but the characteristic feature is a series of irregular, spasmodic, quick movements which are non-rhythmical, and follow each other without any definite order.

The movements, although they are quite independent of volition and take place when the patient is at rest, are increased by trying to perform voluntary actions, while on the other hand voluntary movements are much hampered by the involuntary spasm, and to escape it the patient makes a rush at an object he wants to pick up; emotion or the fact of being watched also increases the movements. Except in very severe cases, the movements cease during sleep. Described more exactly, the upper limbs are suddenly jerked, so that the elbow is flexed and the shoulder adducted and elevated, the fingers and thumb being quickly flexed or extended.

In the early stage the walk is characteristic; one foot appears to be detained, as if it had caught in an obstacle, and then this is overcome, and the patient walks on again quickly as if to make up for lost time, or the feet are thrown about in an inco-ordinated manner.

The face is much distorted by grimaces, the mouth being particularly affected; a peculiar smacking or sucking noise of the lips is very characteristic, and may be one of the earliest symptoms; the tongue, especially when protruded, is moved about in all directions, and is rapidly retracted into the mouth; speech is frequently affected, and varies from mere hesitancy or blurting out the words to an incoherent jumble or inability to say a single word, though they can often cry out apparently involuntarily. In severe cases swallowing may be very difficult or impossible. The muscles of the trunk become affected, causing the patient to rock about while standing up, and the respiratory muscles may act irregularly. The involuntary muscular actions of the intestines, etc., are not involved, nor are the sphincters of the bladder or anus, unless there are mental changes.

Though the movements begin as a rule on one side, they usually involve both sides equally. There are some cases, however, in which the limbs of one side are alone affected, and here it is of great interest to note that the orbicularis oris and the tongue are affected on both sides: these are cases of hemichorea. Muscular weakness, which is not common with chorea except as the result of general feebleness, is most often met with in hemichorea, and may be then associated with loss of sensation—slight hemianæsthesia—on the same side. Hence this hemiplegia may be the most prominent sign, and there may be only just enough movement to make a diagnosis of hemichorea. Sometimes the loss of power is confined to one arm only.

With regard to the knee jerk, in many cases it is not altered; in others it cannot be obtained, probably owing to the muscles being spasmodically contracted. But a peculiar condition which is not obtained in other diseases is that, when on tapping the tendon the knee is extended by a contraction of the extensor cruris, this muscle remains extended for a second or two, and the leg does not fall back at once to the flexed position.

Mental disturbance, as irritability of temper, emotional outbreaks, may occur; or the patient may present distinct mental changes, with hallucinations, delusions, and mania, and it is in these cases that the temperature, which is usually normal, may go up to 104° or 107°.

The condition of the heart is most important in chorea, and excluding the changes which have been produced by a previous attack of rheumatic fever, the conditions arising during an attack of chorea are either functional or organic. Examples of functional symptoms are palpitation, disturbance of rhythm, rapid action and pain which is not very common, except when there is endo- or pericarditis in recurring attacks of chorea. Cardiac murmurs may be either functional or organic, and occur—taking all murmurs found in chorea—in about a third of the cases. Functional murmurs are heard as soft, systolic bruits over the pulmonary artery area to the left of the sternum, and are altered by the patient lying down, increasing or disappearing. The cause of the murmurs may be the same as in cases of anæmia, or they may be due to disordered contraction of the musculi papillares, whereby the mitral and tricuspid valves are not completely closed; it is probable that the former explanation is the correct one—*viz.* the anæmic condition of the patient. Organic murmurs due to acute endocarditis may occur without this producing any cardiac discomfort, and sometimes without pyrexia. They may come on while the patient is under observation; the first sound becomes altered and prolonged, and this is followed by a loud blowing murmur having all the characteristics of being caused by vegetations on the mitral valves; these murmurs may persist after the attack of chorea, or may pass away. In nearly all fatal cases of chorea there have been found changes in the valves or pericardium in as many as seventy-five out of eighty cases (Sturges), the mitral valves being most affected, next the aortic, and rarely the tricuspid.

Course.—The disease lasts at least for six weeks, and it may be prolonged to six months or even a year, and the more severe a case is the longer it will last; on the other hand even cases which are not severe may last several months before complete recovery occurs. Cases of chorea, though most of them get well completely, are very liable to a return of the disease. About one-third of the cases have relapses (Gowers), and they recur in from six months to two years after the primary attack; it is in these relapses that organic cardiac disease very frequently occurs. Occasionally the movements persist for years, and especially in adults. The number of fatal cases is given by different authors at from 2 to 3 per cent., and in uncomplicated cases it is due to exhaustion and inability to take food.

Pathology.—Chorea was formerly considered to be due to minute embolisms, which blocked up the arteries in the basal ganglia, interfering with their nutrition, and thus producing the movements. At the present time it cannot be said that any lesions characteristic of chorea can be found in the nervous system. Hyperæmia, exudations round the vessels and in their sheaths, small hæmorrhages, and emboli have been described in the optic thalamus and basal ganglia, and changes in the nerve cells of the cortex and basal ganglia; but they are too inconstant to be looked upon as causes of the disease. With regard to the situation of the disturbance, it is probably in the cortex, from the fact that in hemichorea, while the limbs are alone affected on one side, the face and tongue movements are observed on both sides. This condition could only be brought about by a cerebral lesion; and as we know nothing about the motor properties of the basal ganglia, and as by stimulating the cortex electrically the limbs of only one side can be made to contract while both sides of the tongue and of some of the muscles of the face can be thrown into action, the lesion is probably in the cells of the motor cortex. As to why these cells should take on this irregular action, and why chorea should be associated with acute rheumatism, we must fall back on the hypothesis, first that the cells of the cortex are in an unstable condition due to the age, sex, and predisposing neurotic history of the patient; and secondly that a particular morbid condition of the blood exists, allied to that which produces acute rheumatism, and "according to the precise nature of the blood change, chorea, or acute rheumatism, or both, with or without endocarditis, may be produced" (Gowers). The result is that the nutrition of the cortical cells is interfered with, and they, so to say, "run riot."

Diagnosis of a fully developed case presents no difficulty. In slight cases making the patient stand with the arms held in front, or over the head, and the fingers separated, with the tongue protruded, will usually bring out any movements, while the peculiar sucking noise is very characteristic. Where weakness of one arm or one arm and leg has come on gradually, there will usually be some slight movements, or a previous history of them sufficient to make a diagnosis.

Prognosis.—In most cases this is favourable, and the patient recovers. Complications such as organic heart disease, mania, and pregnancy make the prognosis worse; and great restlessness producing inability to swallow and to sleep and great weakness are unfavourable.

Treatment.—The most important matter is to procure complete rest, both bodily and physical. For this purpose a child should be taken away from school at once and kept at home; and it is best to keep them in bed until the movements cease or are much diminished, especially as it is quite impossible to foretell the advent of any cardiac lesion. The child should see very few people; but to prevent it from being depressed, it should be read to and amused. In severe cases where the movements are violent the mattress, which should be very soft, should be put on the floor and fenced round with bolsters; if in spite of this the limbs are bruised, the joints should be encased in cotton wool, and if sores form a water bed is necessary.

The question of diet is of the greatest importance, as the fatal cases seem to die from exhaustion consequent apparently from not being able to take food, and milk, eggs, soups, and broth should be given. Osler recommends an ordinary baby's bottle with a flexible tube and nipple when the movements of the head are very marked, but in cases where the patient cannot swallow nutrient enemata or nasal feeding by a soft tube are necessary. Stimulants are required if the pulse fails. The skin is sometimes dry, and a warm bath is grateful to the patient and diminishes the movements for the time, or a hot-air bath may be used.

With regard to medicines, these may be divided into tonics and sedatives. Of the former, iron in the form of the tartrate or carbonate is indicated if there is anæmia. Arsenic has been looked upon as the chief drug in chorea, and it certainly seems to succeed in many cases. It is best given as the liquor, beginning with 2 minims three times a day after meals, and increasing up to 10 minims; but in full doses it should be carefully watched, and stopped if there is diarrhœa, vomiting, itching of the eyelids, pigmentation of the skin, for fear of producing neuritis.

Zinc oxide and sulphate have been useful in some cases, beginning with gr. i to ij, and pushing it as far as the patient will stand without producing vomiting.

Quinine in large doses has been recommended by H. C. Wood; and salicylate of soda is indicated where there is a history of rheumatism.

Sedatives as the bromides and chloral hydrate are used to procure sleep, also antipyrin in 20 to 30 grains in the course of the day, exalgine, sulphonal, and morphia, which should be used very carefully if there is much excitement. Chronic cases in which the movements are severe have been treated by Bastian and others by giving chloral in doses of gr. xx about every six hours; on waking up the patient is fed, and in half an hour's time another dose is given, so that with the exception of his meals the patient is kept always asleep.

In chronic cases strychnine is recommended, and tepid douches to the spine. Electricity in the form of passing a weak constant current through the most affected limbs from the opposite cortex has been used; but it is very doubtful whether any good results.

PARALYSIS AGITANS (SHAKING PALSY, PARKINSON'S DISEASE).

This is a slowly progressing disease occurring after forty years of age, and characterised by rhythmical tremors, general rigidity, with typical expression and attitude. It was first described by Parkinson in 1817.

Causes.—*Predisposing.* *Heredity* can seldom be traced in the form of other nervous diseases in the family.

Age.—It is distinctly a disease of middle life, rarely beginning before forty, and only in isolated cases occurring before thirty. Most cases probably begin between forty-five and fifty-five, but after sixty-five it is rare.

Sex.—Both sexes are liable to be affected, but men more frequently than women.

Syphilis does not seem to be a predisposing cause.

Direct.—The two chief direct causes are mental worry and anxiety, and mental shock or physical injury, as in the case related by Sir W. Gowers, and which I also saw, of a woman carrying a child, and going to the funeral of a friend killed in a railway accident, where the movements came on in the tired arm ; or in one of my cases in which the tremor came on in a finger in which a needle had been broken.

Symptoms.—The disease may be described in three stages. One of the earliest symptoms, and one by which a diagnosis can often be made, as the patient walks into the room, is the general fixity of the expression of the face and the general carriage of the body. In the face there is a certain want of expression, and especially a want of movement in the upper part of the face, when the person speaks, so that if the mouth be covered over it would not be possible from the rest of the face to know that the patient was speaking, while at the same time the speech tends to be monotonous and expressionless. The patient carries himself stiffly, and in turning round in walking the body moves as a whole, and not the shoulders before the hips ; a slight stooping is also noticed, and owing to stiffness at the back of the neck the patient cannot look upwards with comfort. The gait may be unaltered, or be slow and gliding in character. With regard to the limbs, there is no wasting of muscles ; but tremor, which has given the disease its name, is present often as an early symptom. This is a rhythmical to-and-fro movement, affecting usually one hand first, and consisting of a rolling of the thumb and forefinger over each other, or a flexion and extension of the wrist, or pronation and supination of the forearm. The rate of the tremor is from four to eight complete vibrations a second, its great characteristic being that it persists during rest, and only stops when the patient sleeps. It is usually diminished by voluntary action, so much so that patients with marked movement can thread a needle. There is no paralysis at first and no incoordination, but sometimes a difficulty in performing fine movements with the affected hand.

The order of invasion of the tremor is usually of the hemiplegic type, the leg of the same side being involved, then the arm of the opposite side, followed by the leg of that side. Sometimes the arm or leg of the opposite side is involved before the leg of the same side, and occasionally the movement starts in one foot.

Later on the hand has a characteristic attitude, the fingers are extended in the phalangeal joints, and flexed at the metacarpal joints, giving the appearance of a hand holding a pen.

The deep reflexes are as a rule unaltered.

The first stage lasts from two to five years.

In the second stage all the above symptoms are increased, the attitude of the patient is still more characteristic, the head is bent forward, the back rounded, the arms kept a little way from the side, the elbows flexed, the hands carried just in front of the patient, the position of the fingers is accentuated, and they tend towards the ulnar border, while the thumb is pressed against the forefinger and its dorsal aspect becomes concave.

The gait becomes altered, and the patient takes shorter steps. The solidified aspect is well seen as the patient turns round in walking, and also in the great difficulty he has in getting up from a chair.

Another symptom is soon noticed—*viz.* a delay before a voluntary movement is started, and an extreme slowness in its execution. It is this which gives rise to the symptom of running backwards and forwards, called retropulsion and propulsion. Retropulsion is nearly always the first to be developed, and occurs spontaneously, or can be brought about by making the

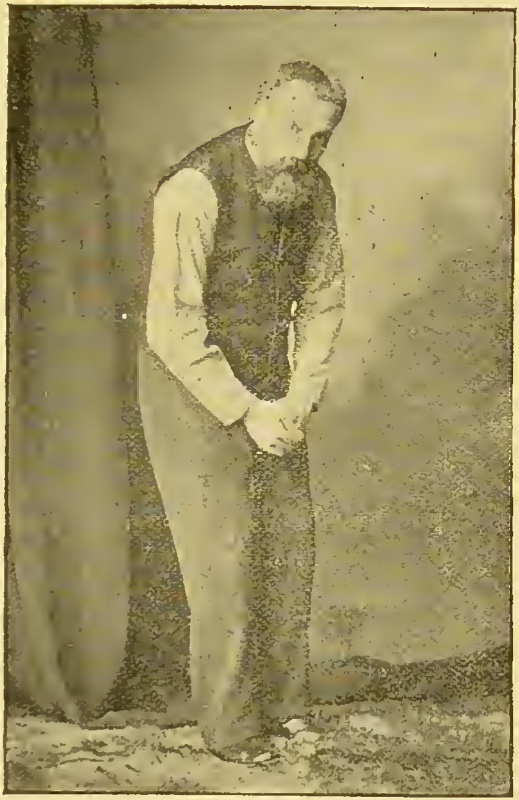

Fig. 28.—Paralysis agitans, in the second stage, showing the stooping attitude, the position of the hands and arms, and the turning of the eyes and not of the head on looking sideways.

patient look upwards, or by gently pulling him from behind, when he begins to run backwards; his centre of gravity is displaced beyond his base, and he is prevented by the slowness of his movements from getting behind it; he therefore has to try and keep up with his body, which is falling backwards. The slow voluntary movements are often well shown by asking the patient to look to one side, when the eyes will turn first, followed by the movement of the head, instead of both being simultaneous.

The tremor by this time has affected both hands or forearms, and to a less extent both legs, the toes and ankles being most affected. The head, tongue, and chin are very rarely the seat of tremor, and the eyeballs never. The writing is characteristic, the up-strokes being tremulous and the down-strokes firm. The difficulty of performing fine movements increases; but there is often no paralysis of force till the end of this stage. The speech becomes, in addition to monotony, high pitched and almost a whisper, as if the patient could not exert himself.

This second stage may last from ten to twenty years.

In the last stage the patient is confined to his bed, becoming very helpless, and at the same time very restless, so that he constantly wants his position altered, and a sense of heat is also complained of. The tremor and other symptoms persist, and general exhaustion with mental obscurity ensues, followed by involuntary passage of excreta, bed sores, and death. The small muscles of the hands are atrophied, and the joints of the hand become fixed.

Pathology.—This is not known for certain, but the most likely site of the lesion is either the cortex cerebri or the basal ganglia, and the changes are probably degenerative, similar to the senile condition. It is to be remarked that the emotional movements of expression are lost, and the automatic movements of the limbs are slow; but in the early stages the patient can hurry the movements when asked to do so. These symptoms would perhaps place the disease rather in the basal ganglia than in the cortex.

Diagnosis.—This is not difficult in well-marked cases, but in the early stages tremor alone is not sufficient to make a diagnosis. On the other hand a fixed expression, a set attitude, monotonous speech, and slow voluntary movements can determine a diagnosis without any tremor. A case was under my care for ten years with all the symptoms of paralysis agitans, and without any tremor except very slight in the chin and tongue.

From senile tremors, which begin in both hands at the same time, affect the head, and are very much increased by voluntary effort, the diagnosis is made by the absence of the fixed attitude, the gait, and the slow voluntary movements. Hysterical tremor occurring in a middle-aged man is differentiated by the absence of the other symptoms of paralysis agitans. Disseminated sclerosis begins much younger, about puberty; the tremor occurs *only* on voluntary movement, is more irregular, and there is nystagmus.

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Prognosis is very unfavourable as regards recovery, but not as regards life. When once the diagnosis is definite, recovery is not to be looked for, though it may be many years before the person becomes helpless.

Treatment.—The patient should live as quietly as possible, and all unnecessary anxiety and worry should be avoided; but if a patient's occupation is not laborious, and not of an anxious character, it is best to continue it as long as possible.

No drugs have been found to cure the disease. In the early stages tonics as quinine and iron, arsenic, strychnine, and phosphorus gr. $\frac{1}{30}$ ter die post cib. in pill, or as the sodic hypophosphite gr. v, have been used; but the first three are the best.

Sedatives, as cannabis Indica, morphia, hyoscyamine, hyoscine, and atropine, are able to control the movement, but have no effect on the disease; and if the tremor is not very severe, it is better not to try and stop it. If the patient is very restless at night, the bromides alone or with chloral, and sulphonal, are indicated.

Warm baths and gentle massage of the muscles have been recommended to relieve the rigidity, but the effect would only be temporary.

With regard to electricity, the constant current has been employed, and also the faradic wire brush; but no permanent improvement has occurred in the cases under my observation.

TORTICOLLIS, OR WRYNECK.

Two varieties of this are met with, the fixed and the spasmodic. The former is in most cases congenital, and depending on injury at birth to one sterno-mastoid rather than to any disease of the nervous system. It will not be further discussed.

Spasmodic Torticollis.

This consists of a contraction of the sterno-mastoid with other muscles of one side, so that the head is continually being rotated with the face turned to the opposite side. The spasm may be tonic, or clonic and intermittent.

Causes.—*Heredity.* In many cases there is a family history of other mental or nervous diseases; and with regard to the patients themselves, they have often had some functional nervous disease. One case I have seen complained of being always "nervous," and objecting to go into crowded places.

Age and sex.—It is a disease of middle life, between twenty-five and fifty, and it is more common among women than men.

As *direct* causes may be mentioned worrying or depressing work, injuries as blows or wrenches of the neck, and catching cold. In one of my cases it was referred to malaria.

Some cases are considered to be hysterical, but it is very difficult to decide.

Symptoms.—As a rule the motor spasm is the first symptom complained of, and it usually begins gradually, but in one of my cases it started suddenly while in church. The spasm is usually attended, and sometimes preceded, by a dull aching pain, or sometimes acute, in the neck, over the affected sterno-mastoid.

The muscles most affected are the sterno-mastoid of one side, most frequently the right (Gowers), the action of which is to draw the head down to the shoulder and to turn the face towards the opposite shoulder:

the part of the trapezius between the skull and the clavicle, which draws the head towards the shoulder of the same side : and the splenius capitis, which draws the head backwards, and rotates the face towards the same side. The usual combination is the sterno-mastoid of one side, the trapezius of that side, and the splenius of the opposite side. These acting together will turn the face to the opposite side with the chin raised, and the head slightly drawn down to the shoulder of the same side. In other cases the sterno-mastoid is alone affected. The rectus posticus and obliquus inferior capitis may be involved.

The spasm may be of a fixed tonic character, or it may be clonic, the head being rotated round to one side, and then swinging back again, and again being rotated in an irregular rhythm. In other cases the head is drawn backwards by the action of the muscles of both sides.

The spasm sometimes involves one of the upper limbs, and the arm which is affected is on the side opposite to the sterno-mastoid. The reason of this is that in health in using the right arm the head turns to the right side to direct it, hence in disease this physiological combination also holds good ; if the head is turned to the right by spasm of the left sterno-mastoid, it is the right arm which is affected. In all the cases which I have seen the humerus has been strongly adducted and rotated in, the elbow extended, and the wrist and fingers only slightly affected. In one of these patients the spasm was attended by very intense pain.

Prognosis and course are unfavourable unless the spasm is of a "hysterical" nature, when patients may recover completely, as in the case of a girl aged seven years, in whom the spasm affected the head and arm and lasted ten years. In most cases the spasm increases up to a certain point, and there remains stationary, or it may in exceptional cases diminish.

Pathology.—An intermittent clonic spasm like torticollis cannot well be produced by a lesion of a muscle or of a nerve, and it can only be due to a lesion of grey matter; and the two most likely places are the cells of the nucleus of the spinal accessory nerve in the medulla and the cells of the opposite cortex cerebri. It seems possible that, when one sterno-mastoid and trapezius of the same side are involved, the lesion may be in the medulla, but when in addition some muscles of the opposite side are involved there is reason to believe that it must be in the cortex. In one case I have seen, the arm was alone affected by clonic spasm after a compound fracture of the skull from a blow on the head; although no gross change was found on the surface of the cortex on trephining, it seems quite possible that the effect of a blow might alter the nutrition of the cells and interfere with their proper action.

Diagnosis of a well-marked case is not difficult. From the hysterical form the age and sex of the patient are the only guide; from spinal caries of the cervical vertebræ where the head is on one side, it will be found that the deformity is due, not to the contraction of the sterno-mastoid, but to changes in the bones.

Treatment.—The patient should lead a healthy life, and avoid worry and trouble, especially if these have seemed to be the starting-point of the illness, and in these cases it is a question as to whether Weir-

Mitchell treatment, described under "Hysteria," should not be given a trial.

Tonics as iron, quinine, strychnine, arsenic, or valerianate of zinc and assafœtida (Gowers) should be given.

Sedatives, unless the pain is very severe, are of doubtful use, for though the spasm can be diminished by opium, morphia, Indian hemp, conium, it frequently returns when they are discontinued. Hypodermic injections of morphia, systematically given, have been recommended; but there is a great chance of the remedy being worse than the disease.

Electricity in the form of the constant current, with the negative pole to the suboccipital region and the positive over the affected muscle, with as strong a current as the patient can comfortably bear, should be used; the current should be increased from 0 without any break, and applied for from five to ten minutes every day; it frequently diminishes the spasm, but as a rule the effect is only temporary.

In cases which are not benefited by medical treatment, a surgical operation should be recommended; and two different operations are to be considered—(1) on the spinal accessory nerve, (2) on the cortex cerebri.

(1) If the spasm is confined to the sterno-mastoid and trapezius of one side, division and excision of from a half to one inch of the spinal accessory nerve, and turning the ends away from each other to prevent reunion, will stop the movement; and if the deeper muscles are involved, division of the posterior branches of the spinal nerves supplying them has been recommended and used with success (Ballance, Keen).

(2) In cases where the spasm involves the arm as well as the head, the above operation would not be sufficient, and the question of operating on the cortex would have to be considered. In these cases, especially if there is much pain, it would be justifiable to trephine, open the dura, and expose the cortex on the side opposite to that to which the face turns, and after finding the exact spot by faradising with aseptic platinum electrodes the cortex, where the particular movement is localised, as recommended by Horsley, to remove that part. In the monkey the exact spot, where these movements of the head and shoulder are represented together, is in the posterior part of the middle frontal convolution just above the præcentral sulcus (fig. 8).

Where patients object to an operation, considerable relief may be obtained from a mechanical rest in the form of a steel rod passing from a fixed support round the chest up the spine to the occipital region, and with a kind of horizontal crutch extending to the mastoid process on either side, and fitted to the skull, against which the patient can lean back the head, and so arrest the movement; but an absolutely rigid position of the head is insupportable.

CHAPTER XXIV.

FUNCTIONAL DISEASES (*continued*).

TETANY.

THIS is a name given to a tonic spasm of the extremities, lasting a few minutes or hours, or even longer, and frequently associated with rickets. It has nothing to do with tetanus.

Causes.—*Predisposing. Hereditary.* There is a history sometimes of other nervous diseases in other members of the family, or other children have suffered from tetany.

Age and sex.—Children under three years are most commonly attacked, but young adults of both sexes are not exempt, and it has been especially noticed in women who are nursing (Trousseau). It also occasionally occurs after forty years, but unusually after fifty.

Rickets is looked upon as a predisposing cause, and tetany is often associated in these cases with laryngismus stridulus.

Exciting causes are a chill, or exhaustion from overfatigue or from wasting diseases, or some reflex irritation, as derangement of the gastro-intestinal canal, diarrhœa, or worms. Surgical operations, especially complete removal of the thyroid gland, and also some of the acute febrile diseases, have been followed by tetany.

Symptoms begin rather suddenly, the first symptom in adults being numbness and tingling in the fingers of both sides, rarely of one side only. This is followed by a tonic contraction of the fingers and thumb; the fingers assume what is known as the interosseal position, due to spasm of the interossei muscles—*i.e.* they are extended at the phalangeal joints and slightly flexed at the metacarpo-phalangeal joints, and adducted together, while the thumb is flexed at the terminal joints, and adducted into the palm under the fingers, which are sometimes flexed over it; subsequently the wrists and elbows are slightly flexed, and the lower limbs are also affected, the toes being adducted and flexed, and the position of the foot being that of talipes equino-varus, while the knees are flexed or extended. The upper and lower limbs may be alternately affected, but rarely the lower limbs alone. Occasionally the muscles of the eyeballs, the face, the tongue, and the lower jaw are affected with tonic spasm, and less frequently those of the trunk and thorax; but even the diaphragm has been described as being affected.

The spasm is tonic, and can be overcome by passive movement, but the joint resumes its original position when left to itself. In slight cases there is no pain, but in severe cases cramps are felt in the muscles. The spasm lasts from a few minutes to a few hours or longer, and then passes off to again recur. In slight cases the patient can with difficulty overcome the spasm voluntarily, but not in severe cases.

There is usually no affection of sensation in the parts which suffer from numbness, but occasionally there may be partial loss to tactile or painful impressions.

Between the attacks of spasm a most curious hyperexcitability is observed in the nerves, so that very slight pressure or tapping on a nerve produces contraction of the muscles supplied by that nerve (Trousseau), and if continued it produces a prolonged contraction. The cutaneous reflexes are increased, but not as a rule the knee jerk.

The electrical reactions of the nerves to the induced and to the constant current are much increased, and also the mode of reaction (p. 73) to the constant current is altered (Erb), so that the nerves react first to the positive pole (anode) when the circuit is closed, and next to the anode when the circuit is broken or opened, and lastly contract to the negative (cathodal) closing current; also a prolonged contraction or tetanus is produced by a momentary application of the poles instead of a single contraction, even with the anodal opening (A.O.C.). The order of contraction is therefore A.C.C., A.O.C., and K.C.C., in place of K.C.C., A.C.C., A.O.C. of health.

As a rule there are no constitutional symptoms, but in severe attacks the temperature may be raised, with redness and œdema of the skin and profuse sweating.

Course and variety.—In some cases, especially in children, the spasm is continuous, and then passes off after several hours or days and does not recur, while in adults the spasm lasts a shorter time and recurs, so that the whole duration is longer in the latter cases.

Recovery takes place in the majority of patients. In a few cases death has been due to the spasm or to the pneumonia induced by the contraction of the respiratory muscles producing deficient aeration (Trousseau).

Pathology of the disease is still obscure; but it is probably a deranged condition of the cells of the grey matter of the medulla and spinal cord, and so of the nerves supplied by these cells; and whereas in epilepsy the cells of the cerebral cortex when hyper-excitable produce tonic followed by clonic contractions, the motor cells of the spinal cord produce only a clonic contraction. Whether this state is brought about by some toxic condition, or whether a poison is produced within the system, is a matter of conjecture.

Diagnosis has to be made from tetanus, in which the spasms are more violent, and affect the muscles of mastication early. From hysterical contracture it is often difficult and sometimes impossible, but in the former there is not the hyper-excitability of the nerves, and in the altered electrical reactions there is no other disease besides tetany which gives a prolonged contraction to the anode when the current is opened.

Prognosis, except in very severe cases, where the spasms are violent and interfere with respiration, is good; but the patient is liable to a recurrence. It is quite impossible to say how long an attack will last; but the cases in which intermission does not occur usually last a shorter time than those which intermit, where the disease may be prolonged for several months.

Treatment.—This consists firstly in removing the cause, secondly in arresting the spasm by internal or external remedies, and lastly in improving the general health. In the first place irritating causes, as diarrhœa or worms, must be inquired into and treated. In arresting the spasm, Trousseau employed bleeding from the arm and cupping over the spine when the patient was of a vigorous constitution and there was marked febrile reaction, in other cases he gave quinine with opium or belladonna.

The spasm is best relieved by the bromides in gr. xxx to xl twice or three times a day alone or with chloral or cannabis Indica, and if the spasm be violent chloroform inhalations are required. Local applications to the affected muscles of chloroform on a piece of lint with ♏v taken internally every hour have been used (Aran). Electrical treatment in the form of the constant current, with the negative pole on the spine and the positive applied to the muscles in the stabile method, without breaking the current relieves some cases (see p. 73).

The general health should be improved between the attacks by cod-liver oil and tonics as strychnine, arsenic, iron, and quinine; and in the case of children the presence of rickets should not be overlooked as a cause to be treated.

CHAPTER XXV.

FUNCTIONAL DISEASES (*continued*).

OCCUPATION SPASMS: WRITER'S CRAMP.

THIS disease, which was first described by Sir Charles Bell in 1830, is a type of the spasmodic condition which may affect muscles which are frequently used for some definite class of movements. Every occupation which entails repeated movements of the same kind, as in writing, has its form of spasm; there is therefore violin and pianoforte cramp, seamstress' cramp, sawyer's and treadler's cramp, etc.

Causes.—Predisposing are usually present. The patient is often of a distinctly "nervous type," and may have suffered from migraine or neuralgia. *Hereditary* predisposition occurs, as epilepsy or insanity, in other members of the family.

The age is that of adult life, between thirty and forty especially, the disease not occurring in very young or old people. More men than women are affected; but this is probably due to more men being engaged in these occupations.

Exciting causes are the excessive use of certain muscles in one particular set of movements, and a condition of much anxiety and worry. Injury to the hand has been considered in some cases to start the condition.

The class of people who suffer most are those who have to write not only for several hours a day, but in a definite style of writing, *i.e.* copperplate, which entails a constrained position of the hand; hence a very large proportion of the cases are lawyers' clerks, while the disease is practically unknown among shorthand writers, who use a very free style (Gowers). Writing as taught in schools is performed by the movements of the small muscles of the thumb and the interossei of the first two fingers, which have not only to hold the pen, but also to direct its movements. Further, in forming the letters the distal phalangeal joints are extended, while the proximal are flexed. The hand has to be balanced on the tip of the little finger, or on this and the wrist, and the forearm is pronated in a constrained position. The fact that most people, who are not obliged to write copperplate, give up the style which they have learned at school is strong evidence of the method being quite wrong, and most people when left to themselves hold the pen in the way which comes easiest to them. For those who have to do excessive writing, the less the fingers and thumb have to do with the movements for the formation of letters the better, and Sir W. Gowers advises that this should be done by the muscles of the shoulders, the hand and forearm moving as a whole. Probably the most natural way is to hold the pen as an artist holds his pencil.

Symptoms.—These may be either sensory or motor, or both, motor being the more common. The patient complains that gradually, after writing a certain time, there is pain or a tired feeling, beginning in the small muscles of the hand, and then extending up the arm sometimes to the shoulder, or a feeling of pins and needles is experienced. At the same time there is a difficulty in holding the pen, which slips away from the fingers, and has consequently to be held tighter. After a time there is a distinct difficulty in making the pen move in the direction required, the movement of the hand is prevented by a tonic spasm of its muscles, or in other cases there is a spasmodic contraction of the muscles by which the forearm is suddenly supinated and the pen thrown off the paper. The writing gradually becomes very scratchy and tremulous, and at last impossible. The patient tries to get over his difficulty by grasping the pen with the whole hand, or holding it between the first and second fingers, but with the result that the muscles of the upper arm and shoulder become affected. At first the pain and spasm cease as soon as the writing is left off, but after a time the pain persists, and there is tenderness over the nerve trunks of the limb, especially the median, ulnar, and musculo-spiral nerves, presenting what is known as the neuralgic type.

In most cases the patient can use the muscles to perform any other movement except the one involved, even those requiring delicate adjustment of the muscles. There is no loss of power, or perhaps slight weakness of certain muscles of the hand, no wasting of the muscles, and no anæsthesia.

As a rule the electric reactions of the muscles and nerves are normal, but Sir W. Gowers has described increased irritability, followed by diminution to both faradic and galvanic currents.

Course.—If the particular occupation, *e.g.* writing, which causes the spasm be persevered in, the affection increases, until at last it is impossible to take the pen in the hand without causing a spasm, and writing becomes impracticable. If however writing be completely discontinued, the spasm tends to diminish and may entirely disappear, but it is liable to return on resuming writing. Patients often use the left hand for writing, and in a certain proportion of the cases this becomes affected with the spasm.

Pathology.—No lesion has ever been found in the nervous system; but three theories have been propounded to explain the condition: (1) that it is due to local paralysis or weakness of certain muscles, causing overaction or spasm of their antagonists; (2) that it is a reflex spasm produced by hyper-excitability of the sensory nerves; (3) that it is a functional derangement of the central nervous system. The first theory is strongly supported by Poore,[1] but against it is, that the derangement of the movements is far more complicated than could be explained by a local paralysis, which often cannot be found; while in the second or reflex theory sensory symptoms do not always exist or precede the motor. The third theory is probably the correct one. The co-ordination of movements required for writing has to be learned in childhood with great difficulty, but after a time by practice they become automatic.

In writing only certain muscles are used, and the constant and excessive repetition exhausts the motor and sensory cells in the spinal cord (or in the cortex) supplying these muscles, and in consequence they are unable to regulate the muscles in the proper performance of the movements for writing.

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[1] *Med. Chir. Trans.*, vol. lxi.

Diagnosis.—This is not difficult if it be remembered that the spasm and the inco-ordination only occur for one particular occupation, such as writing, and that other actions, even those requiring delicate adjustment, can be performed. There is less chance of overlooking the disease than of thinking that every difficulty of writing, whether caused by disseminated sclerosis, paralysis agitans, etc., is a case of writer's cramp, thus overlooking the main disease.

A spurious form of writer's cramp, or "stammering writing," occurs in nervous people who cannot write when looked at or when flurried. The difference from true writer's cramp is that these cases can write properly and for any length of time when alone, and that if made to write slowly under observation the writing improves the longer they write, whereas in true writer's cramp it begins fairly well and gets worse after a few minutes.

Prognosis is very uncertain. Unless the occupation can be given up it is hopeless ; but when this can be done, and unless there is some mental worry and anxiety to keep up a nervous condition, the chances of cure are fairly favourable ; but the longer the disease has lasted the worse the prognosis.

Treatment.—This consists in absolute rest from the particular action which gives rise to the movement ; in improving the general health by tonics, iron, quinine, strychnine, and cod-liver oil or maltine; and by a complete holiday in the country or at the seaside.

With regard to local treatment, the most efficacious are electrical and massage.

The constant current is applied, with the negative pole to the cervical spine and the positive pole to the muscles of the hand in the stabile (p. 73) manner, using as strong a current as the patient can bear without discomfort. Poore places the positive pole in the axilla and the negative on the ulnar nerve just above the elbow, and uses a current not quite sufficient to cause a muscular contraction, and while the current is passing he makes the patient flex and extend his fingers and exercise the muscles.

Systematic massage to the affected limb, combined with making the patient practise exercises such as flexing, extending, and separating the fingers, is of great service.

For people whose livelihood depends on writing, it is best to give up writing with the right hand and use the left—all the forms of mechanical pens are useless as the fresh muscles which are called into play become affected—or, if the left hand fails, to use a typewriter. When after a course of treatment the patient begins to write again, he should use the method of writing with the whole arm, without moving the fingers or wrist (Gowers), by making a continuous straight line across a sheet of paper, then making this line wavy, and then converting the waves in a third line into *mmm*, and so into other letters. Large cork penholders are the best.

CHAPTER XXVI.

FUNCTIONAL DISEASES (*continued*).

EXOPHTHALMIC GOÎTRE.

THIS is a disease characterised by three main symptoms—prominence of the eyeballs, an enlarged thyroid, and rapid cardiac action. It was completely described by Graves in 1835 and by Basedow in 1840, and is often called after their names.

Causes.—*Hereditary.* In many cases there is a history of epilepsy, paralysis, insanity, or some disease of the nervous system in other members of the family. In rare cases one of the parents has suffered from this disease.

Age and sex.—It affects people in adult life, and most frequently between fifteen and forty, and females very much more than males.

Wasting diseases, and especially anæmia, predispose to the disease, as also do prolonged anxiety and overwork. One of my cases, who was excessively anæmic, ascribed her illness to nursing her mother and then losing her brother rather suddenly.

As *direct causes* a sudden shock, either mental or physical, has seemed to start the symptoms, and also violent exertion, or excesses of any kind.

Symptoms.—In most cases the symptoms begin with a tired feeling, palpitation of the heart, faintness, and shortness of breath. Later on, sometimes after several months, the thyroid begins to enlarge—usually one lateral lobe before the other—and subsequently the eyeballs are noted to be more prominent than natural, although this symptom is sometimes entirely overlooked by the patient and her friends.

When the disease is fully developed, the pulse is regular, but very rapid, being up to 136, or higher, even when the patient is sitting or lying down, and it is readily increased by any mental excitement or physical exertion. The heart soon shows signs of dilatation, the apex is felt more outwards, and the impulse is diffused. There may be no murmur, but only excited action, with loud sharp sounds; while in other cases there is a loud systolic murmur, heard most at the apex, but also at the left base, and in some cases over the carotids and the thyroid gland. The pulsation in the large arteries is often very marked, and may give rise to a thrill.

After the rapidity of the pulse has existed for some months, the goître, which gives the name to the disease, begins to be formed by enlargement of first one lateral lobe of the thyroid, and later on of the other side; the enlargement is without pain, and is accompanied by marked pulsation of the arteries. The enlargement of the thyroid is more marked at the times when the heart's action is increased (Graves).

The protrusion and prominence of the eyes take place about the same time as the goître occurs, but may precede or follow it. One eye is often affected first;

but there appears to be no relation between this and the side of the thyroid first affected. The eyes are noticed to be rather more staring than usual, and this is found to be due to the conjunctiva being visible above and below the iris. This increases until the eyes are distinctly prominent, so that the eyelids are unable to meet, and the corneæ may become dry or inflamed.

Another condition, which has been called Von Graefe's symptom, consists of a lagging behind of the upper eyelid when the patient looks down; the eyeball is directed slowly downwards following the moving finger, and a considerable amount of conjunctiva is seen before the eyelid begins to descend, and then it suddenly overtakes but does not keep pace with the eyeball as the lid lags behind again; or in other cases it does not descend at all. The upper eyelid is occasionally unduly retracted, and this has been called Stellwag's symptom. The pupils are normal.

Of all the symptoms, increased rapidity of the heart's action is the most common, but the disease often remains unrecognised till the other symptoms are added. Another symptom which is met with is tremor, especially of the hands; it may be irregular like chorea, or regular like paralysis agitans, or it resembles the tremor of disseminated sclerosis in that it occurs when a voluntary movement is made.

Patients are frequently anæmic, and they lose flesh. One case, aged forty, had been reduced from nine to less than five stone in weight, and suffered from functional paralysis of the legs. An emotional or irritable mental condition is often present, and this may pass on to actual mania or melancholia.

Other nervous affections, as migraine, epilepsy, paralysis of some of the cranial nerves, and paraplegia have been observed in association with this disease; but the variability of these affections makes it difficult to say whether they are part of the disease under question or merely an expression of a neurotic tendency. Vaso-motor changes occur, such as local œdema, flushing and sweating of the face; while glycosuria, enlargement of the lymphatic glands, and pigmentation of the skin are met with in other cases.

Pathology.—In the orbit there has been found an increase of fat and enlargement of the vessels, with elongation of the optic nerve; in the heart dilatation or hypertrophy, and in some cases valvular disease; the arteries and veins have been found dilated and aneurysmal, this being especially the case in the thyroid gland, which is also found to have its gland tissue hypertrophied. With regard to the nervous system, the vaso-motor conditions have led observers to examine the sympathetic system, but the cervical ganglia have been found healthy or without sufficient change to account for the symptoms; the medulla oblongata has also been found normal.

Taking in consideration the absence of pathological changes, it is probable that the disease is a functional neurosis of the central nervous system, and most likely of the medulla oblongata. This would explain the rapid action of the heart, and its dilatation or hypertrophy would be the sequel to this condition. The enlargement of the thyroid has been considered due to vascular dilatation; but this cannot always be the cause, as the goître has sometimes preceded the rapid cardiac action. The prominence of the eyeballs has also been put down to vascular dilatation and to contraction of the unstriated muscles of Müller, which are supplied by the sympathetic, and are supposed to be able to protrude the eyeball. The non-descent of the upper eyelid is probably due to a functional derangement of the part of the third nucleus for innervation of the levator palpebræ, which in health is gradually relaxed in looking downwards.

The fact that removal of the thyroid produces myxœdema with mental depression, and that injection of thyroid extract increases the pulse rate, has led some observers to refer all the symptoms to an excessive secretion and absorption of thyroid matter.

Diagnosis is not difficult when all the symptoms are present; but in the early stages the presence of a rapid pulse which persists when the patient lies down and when he is free from excitement should make one be careful to watch for other symptoms of the disease.

Prognosis is very uncertain; the earlier the case is seen and the slighter the symptoms the better the chances of recovery. In most cases it is unfavourable, but some cases improve very much, and a few get quite well, though they are liable to a relapse. Prognosis is better when the general health is good and there is no organic heart disease.

Treatment.—Absolute rest and freedom from any mental worry or anxiety are essential, and this can be best carried out by keeping the patient in bed. A sea voyage has been recommended; but one case I saw had just returned from a cruise, and was very emaciated, and worse than before starting. This patient was put under strict Weir-Mitchell treatment for six weeks, followed by a stay at the seaside, and her weight was increased by five and a half stone; she was also treated by tinct. belladonnæ in increasing doses up to ♏xxv with inf. digitalis three times a day, and the constant current applied as described below. In this case the circumference of the neck over the enlarged thyroid was reduced one and a half inches, and the pulse was reduced from 130 to 116; and although the disease was not cured, she expressed herself as feeling quite well, and was able to walk a couple of miles without being tired.

Belladonna, digitalis, and iron are the medicines which give the best results. Trousseau, however, condemns iron as doing harm, and he recommends digitalis and hydropathy, and bleeding if there is danger of asphyxia from pressure, with ice to the præcordial and thyroid regions.

The constant current should be applied for from five to ten minutes every day with a current not strong enough to be uncomfortable—about three milliamperes—the positive pole being applied to the cervical spine, and the negative passed slowly, without being removed, along the anterior edge of the sterno-mastoids alternately. Faradism of the neck has also been employed.

As surgical remedies, excision of the isthmus to produce atrophy of the thyroid, ligature of the thyroid arteries, and tracheotomy are only advisable when there is sufficient enlargement of the thyroid to cause suffocation.

CHAPTER XXVII.

FUNCTIONAL DISEASES (*continued*).

HYSTERIA.

This is a definite disease due to a functional disturbance of the highest cerebral centres, and unattended with any discoverable changes in the nervous system. Hysteria is a very bad term, especially as it means to the laity either deliberate imposture or a condition which patients can prevent if they like.

Indirect causes.—*Heredity* plays a very important part, and in a considerable number of cases there is a history of hysteria or of some other nervous disease in the mother or other members of the family.

Age.—Puberty is the most common age, and most cases occur between fifteen and twenty; but no age is exempt, and cases have been observed as young as six years, and I have seen two cases over sixty.

Sex.—Women are much more affected than men. Twenty years ago it used to be taught that the patient, being a man, excluded the disease being hysteria; but that is certainly not the case now. In France the proportion is one man to every two or three women; but this is certainly too high for this country as regards men. Boys at puberty are especially affected; but one of the cases over sixty I have seen was a man.

Syphilis has no effect except as a general nervous depressant.

Early training in bad surroundings, the example of a hysterical mother, and freedom from any salutâry restraint predispose.

Race.—The Southern (Latin) races are more liable than the Northern (Saxon), and the manifestations are more marked. The Semitic race are especially subject.

Direct causes are physical, mental, and traumatic, which probably acts partly through the mind. The physical causes are bad health, anæmia, or some serious illness. The mental causes are some emotional disturbance, bad news, a fright, a love affair, or loss of money and position ; in other cases an absolute want of aim in life, or too much luxury. Traumatic causes vary from slight inflammation of the eyes or a trifling blow to a severe railway accident. Disorders of the generative organs, as displacement of the uterus, have probably been overrated as causes, but sexual excess is sometimes a cause in men and boys. Another cause is moral contagion, as in institutions where it may act like an epidemic.

Symptoms.—Given an unstable nervous system, due perhaps to hereditary predisposition, any circumstance requiring self-control may bring on hysteria. The simplest case is that of a "nervous" man who has to make a speech in court before a large audience or to appear on the stage as an actor, and fails. Here loss of control in the higher centres, due to the fear of not doing well, produces two sets of symptoms—the lower centres are released from the control of the higher centres, and he has palpitation of the heart or severe perspiration, while, his higher centres being paralysed, he forgets all he has to say or is completely dumb. But the moment after he leaves the court or stage he can speak perfectly well. Here the hysteria is temporary, and may by degrees be overcome; but for the time being he has absolutely no control over himself, and severely resents his condition, which may spoil his chances for life. All the symptoms of hysteria can be compared to this case; but instead of being temporary they are in most cases more persistent. The symptoms may be motor, sensory, vaso-motor, or affect the lower centres of the heart and respiration.

Motor symptoms.—These are due either to diminished action, *i.e.* paralysis, or to overaction of the lower centres producing spasmodic conditions, *i.e.* tremors, convulsions, and contractures.

Paralysis commonly affects both lower limbs, producing paraplegia, or less commonly hemiplegia, or only one limb may be involved; or there may be double ptosis, or paralysis of the vocal cords causing aphonia. The onset may be gradual or sudden.

Hysterical paraplegia often occurs suddenly from some emotion; the paralysis may not be complete, but it is sometimes. The lower limbs are as a rule quite flaccid, the voluntary movements, if present, are performed in a feeble, half-hearted way, and in trying to dorso-flex the ankles a slight attempt, which is improved by encouragement, is made. Some cases can walk shuffling along, others are quite unable to stand, and collapse on the floor at once, when put on their feet. There is no wasting beyond that of disuse, and there is no alteration in the muscles to electrical testing.

The plantar reflexes are often absent, while the knee jerks are normal or slightly increased; there is no true ankle clonus, but sometimes an irregular voluntary clonus is produced by the patient starting the movement by pressing the foot against the observer's hand, but no clonus can be set up by striking the tendo Achillis made tense by passive flexion of the ankle, as in organic cases. Retention of urine may occur, but there is never incontinence.

Hemiplegia is less common than paraplegia. The left side is especially affected, and as a rule the face escapes, though I have seen paralysis of the tongue; the onset is usually gradual. The paralysis is frequently not complete, and is often attended with hemianæsthesia of the same side. The knee jerk is not more active on the paralysed side than on the other, and true ankle clonus is absent. Complete paralysis of one limb may occur with contractures. Paralysis of both vocal cords causing hysterical aphonia is very common, and on laryngoscopic examination the cords are seen to move well, but are not adducted close enough for phonation, though as a rule the patient can cough. Bilateral ptosis occurs, often with overaction of the frontales; but paralysis of the external ocular muscles is exceedingly rare, and when it occurs it is then conjugate.

Over-action of the lower centres produces spasmodic conditions: (1) tonic contractures, (2) clonic movements.

(1) Tonic contractures of the limbs are liable to occur, especially in cases after injury, but also in ordinary hysteria. In one of my cases, after a blow

on the wrist from a lift with the fright of impending death, in two or three days the arm became anæsthetic for all forms of sensation up to the shoulder, and in two more days the arm was completely paralysed and rigid, the hand being firmly closed by a tonic spasm, and resisting all attempts to open it. In another case, of a Russian Jewess, there was contracture of both hands, with complete anæsthesia and loss of voluntary power. The contracture disappears under chloroform or during deep sleep.

One leg is also sometimes affected with extensor spasm, the foot being inverted, and the toes flexed.

Contractures, especially when associated with anæsthesia, may resist every form of treatment, but may finally cease suddenly after some strong emotion. Phantom tumours in the abdomen are caused by spasm of the diaphragm and relaxation of the abdominal walls.

(2) Clonic movements may be in the form of regular or irregular contractions. In the former we have the same movement, as adduction of the arm or rotation of the head, repeated in a more or less regular manner, or, as in another case of mine, elevation of the ears; or the tremor is general, and so fine as hardly to be seen, or violent enough to shake the whole patient.

Convulsive attacks, to which the names of hysteroid fits or hystero-epilepsy have been given, frequently occur, but they are much less violent in this country than abroad, where they have very marked phases. Here the patient has feelings of malaise or tingling sensations in both feet, or feels giddy, or experiences a lump in the throat—globus hystericus. She falls down suddenly without hurting herself and without change of colour in the face, becomes rigid, the arms are abducted horizontally (the cruciform position) or kept close to the trunk with the elbows extended, the hands are clenched tight, the eyelids closed and resist being opened, and when they are forcibly opened the eyeballs turn upwards; the rigidity then changes into a fine general tremor, which becomes more marked, and passes on to the violent stage, in which the patient rolls over and over, or shakes her head from side to side and bangs it down on the floor by violent extension of the neck; it is in this stage that the "grand movements" occur, and the spine is strongly arched concave backwards, giving rise to opisthotonos so that the patient rests on her head and heels; grimaces and convergent squinting occur, but never divergent strabismus; violent purposive movements are made, the patient pushing away people holding her, and trying to bite them; the patient then gives a scream and lies quite quiet, with perhaps a slight flicker of the eyelids, and after a minute becomes rigid again with tremors, or passes at once into the violent stage, or recovers.

Cases may bite their lips or their own fingers, but not their tongues; they do not pass their water involuntarily, the pupils react to light, and the conjunctival reflex is usually retained, and by blowing in their faces suddenly a reflex blinking is produced; they may scream at intervals during the attack, and talk nonsense or sing. The strength which these cases exhibit in the violent stage is quite extraordinary. The suddenness with which they pass out of the attacks is very characteristic and quite different from true epileptic fits. Consciousness in the highest centres seems undoubtedly lost, and although purposive actions are performed the patient has no recollection of them afterwards; on the other hand, consciousness can usually be restored by strong stimuli.

Sensory symptoms.—Disturbances of sensation are very common, and occur in the form of excessive sensibility or hyperæsthesia, and diminished sensation, as anæsthesia, analgesia.

Hyperæsthesia is particularly met with along the spine, especially about the upper dorsal and lumbar regions, and affects the skin as well as the deeper structures, in the infra-mammary, hypochondriac, and iliac regions, especially on the left side; it also involves the special senses, causing intolerance of light and noises in the ears. Anæsthesia and analgesia, with loss of muscular sense and loss to heat and cold, may affect the whole of one-half of the head, limbs, and trunk, with the mucous membranes of that side; the hemianæsthesia is most frequently on the left side, and is sharply limited in front by the median line of the head, face, chest, abdomen, and behind by the spine. Associated with this anæsthesia there is diminution of sight of the eye of the same side, with the field of vision limited equally in every direction to 5° or 10° round the centre of vision, and sometimes there is complete loss of colours, or achromatopsia (often unknown to the patient), so that all colours are called varying shades of grey or black; while in the opposite eye there is a slight limitation of the field of vision to 20° (in place of 90°). Hearing, taste, and smell may also be lost on the same side as the anæsthesia. This condition of vision, called "crossed amblyopia," occurs much oftener than is usually supposed, and can often be found when looked for. Loss of all forms of sensation may occur in cases where one arm is the seat of contracture, and up to where the limb joins the trunk sensation is lost in the whole arm to the actual cautery and to the strongest faradic currents. Loss of sensation in both legs associated with paraplegia is not so common.

Visceral and vaso-motor symptoms occur, as dysphagia, severe vomiting with hæmatemesis, giving rise to "fasting girls"; palpitation, and a sinking feeling at the epigastrium; rapid respiration from 50 to 80 a minute, laryngeal spasm, a barking brassy cough, or persistent hiccough lasting for months, or hysterical hyperpyrexia.

Mental conditions, ranging from excessive emotion—laughing and crying—to actual insanity, are met with.

As rare conditions associated with hysteria may be mentioned the hypnotic condition, and trance or catalepsy. Two cases I have seen who could not look at an object for a few seconds without becoming hypnotic or cataleptic. The patient in the hypnotic state looks as if asleep, the reflexes are present, and she will often answer questions and obey commands, but not with the same promptitude as in a person purposely hypnotised. In the cataleptic condition the peculiarity consists in the limbs remaining for a long time in the position they are placed, *i.e.* the arm remains horizontally extended, and the unconsciousness is often very profound with loss of the reflexes, only the pulse and respiration showing that the patient is alive; and they may die in this condition.

Pathology.—No changes have hitherto been found on post-mortem examination, and from the sudden way in which cases recover it is probable that there are no organic changes ; but hysteria may pass on to, or may be the first stage of, organic disease—*i.e.* disseminated sclerosis. Uncomplicated hysteria is a pure functional disease of the highest centres, and the symptoms may completely pass away. It is considered by some observers that the lowest centres, *i.e.* of the medulla and spinal cord, are primarily affected ; but it is more probable that they are under the influence of the highest centres, which are the parts at fault ; the vaso-motor centres presiding over blushing and secretion of urine are known to be affected by the condition of the highest centres, as many students at an examination experience.

Diagnosis is often very difficult, and can sometimes only be made by excluding all known organic diseases ; and it must be remembered that hysteria and organic symptoms may coexist. Hysteria has to be diagnosed, on the one hand, from organic disease, and on the other from malingering or wilful imposture. Hysterical paralysis affecting the legs has to be diagnosed from myelitis by the absence of incontinence of urine, by the absence of ankle clonus, and often by inconsistencies —one case could not extend the knee, but when it was extended for him and he was told to keep it straight he could prevent forcible flexion by using the same muscles which for another purpose he was unable to put into action—and by the absence of the plantar reflexes. Normal reaction of muscles to electrical testing and absence of wasting are not in favour of hysteria, as they may be met with in myelitis of the dorsal region of the cord; but changes and loss to electrical testing prove organic disease. Hysterical anæsthesia is often difficult to separate from the organic form; but, when it affects one limb only, it is usually associated with hysterical contracture of the limb, and when in the form of hemianæsthesia and the mucous membranes are involved, it will often be found that *both* sides of the uvula and pharynx are anæsthetic and that there is limitation of the visual fields with loss of colours on the same side, and if associated with hemiplegia the face usually escapes paralysis and the deep reflexes are not increased on that side. Hysterical contracture differs from the late rigidity in hemiplegia (p. 234) in being an active spasm—*i.e.* the closed hand involuntarily resists being opened, and the springlike closure of the fingers is increased by forcibly opening the hand. Tremors, especially of one arm, can often be traced to a fright or blow; but when they are general and occur only on voluntary action, the absence of nystagmus is the chief diagnostic symptom from disseminated sclerosis. In hysteria the tremors often persist while the patient is lying down at rest, in disseminated sclerosis never. With regard to the diagnosis from epileptic fits, see p. 298. Wilful malingering can usually be detected by repeated examination. Age and sex are very misleading aids. Twenty years ago it was taught that the male sex and advanced age excluded hysteria; but this certainly cannot be said now. Both disseminated sclerosis and hysteria are very liable to occur in girls at puberty.

Prognosis is, as a rule, favourable, though some cases resist treatment for a long time. The danger of a relapse or the starting of fresh hysterical symptoms is very common from worry or an accident; but if the patient can be put under healthy surroundings, it is quite possible to recover completely. On the other hand, some cases never completely recover, and their symptoms are protean.

Treatment consists essentially of moral and physical means to restore the patient's deficient will power. All exciting causes must be removed, and especially the unhealthy surroundings of home life, which may be uncomfortable from worries or too comfortable from sympathetic relations. For these reasons it is absolutely essential that the patient should be taken from her friends, and put into private rooms or an institution with a reliable nurse with good tact, and in a great number of cases, especially where there is wasting, the treatment first recommended by Weir Mitchell[1] is most useful. This consists (1) in keeping the patient in bed in complete seclusion from every one, not seeing her friends or having letters or newspapers; (2) having general massage and general faradisation of the muscles every day; and (3) being fed up. Skimmed milk 4 ounces every two hours slowly sipped and increased up to 2 quarts daily was recommended by Weir Mitchell for cases of extreme emaciation and distaste for food, but in many hysterical cases full diet can be taken with 6 pints of milk a day. The patient should be kept in bed for a month or five weeks, then allowed to get up, and in a week's time to go to the seaside for from two to four weeks with the same nurse if possible, and without seeing her relations or friends.

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[1] *Fat and Blood*, by Weir Mitchell.

Local treatment for paralysis, as the faradic wire brush, is very useful; and it should be applied so as to act as a stimulant to the patient's deficient will power. In complete aphonia it is usually sufficient to tell the patient that the current will help her to speak, and on applying the wire brush outside the larynx the involuntary "Ah," produced reflexly, will show the patient that she can speak. Also, in inability to raise the leg off the bed, the sudden application of the wire brush to the calves, with at the same moment telling the patient to make an effort, will enable her to raise the leg and keep it up if the wire brush is then kept an inch off the calf.

For anæsthesia faradisation with the wire brush is efficacious, if it can be felt; but in some cases the strongest currents can be applied without the patient feeling it at all, thereby showing that the cases cannot be malingering. Blisters, or the actual cautery applied circularly to the limbs, succeeds sometimes, and the mention of the actual cautery often acts mentally as a cure. A circular blister to the neck will often cure aphonia. Hypnotism I have tried in two cases of contracture; but in one of traumatic contracture and anæsthesia it failed to do any good at all, and in the other the contracture returned a few hours after recovering from the hypnotism. It is unadvisable in principle to diminish the patient's self-control and put it in the hands of other people.

Contractures which resist faradism and blisters are sometimes cured by electricity from a frictional machine, the patient being connected with one of the conductors while sitting on an insulated stool, and sparks are taken from the affected part by means of another conductor connected with the earth, and the treatment probably acts mentally.

Hysterical fits can sometimes be prevented at the onset by the patient douching her face and head with cold water; during the attack the patient must never be held (friends frequently say that it takes three or four strong men to hold a girl of eighteen), but a pillow should be put under her head on the floor, and all clothes loosened round the neck and chest, and the patient should only be kept away from furniture, as she will roll under a table or knock chairs over. Strong stimuli, as faradisation with the wire brush, pouring cold water on to the face and chest, a strong pinch of snuff, are useful in stopping a fit; or the method may be employed, which was introduced by Dr. C. J. Hare, of producing temporary asphyxia by closing the mouth and nostrils with a towel, and on suddenly letting go the patient takes a long breath and the fit ceases.

Pressure over the ovarian region, though successful in France in arresting an attack, often fails here; but pressure of the supra-orbital nerve against the roof of the obit is efficacious, as is the hypodermic injection of $\frac{1}{16}$ gr. of apomorphia (Gowers), though very unpleasant. Hysterical vomiting is best treated by feeding by nutrient enemata.

Medicines as tinct. valerian or assafœtida seem to act, especially in cases of hysterical fits, when bromides fail. Sedatives as morphia and chloral should be avoided, and the first treatment with many cases is to stop the nightly use of chloral. Sulphonal is not so objectionable, but bromides are the least harmful. General tonics as iron and quinine are necessary in anæmia and loss of appetite.

The great point with hysterical cases is to gain their confidence, and on no account to ridicule their symptoms, and with cases of paralysis not to be in a hurry to go too fast; and though occasionally some cases can get up and walk after one application of faradisation, most cases have to go through the stages of walking with help—and here it is best for the attendant to hold the patient's arm, and gradually diminish the support, rather than for the patient to hold the attendant's arm—and then with a framework on wheels (like a large "baby walker"). The treatment of trance and catalepsy consists in feeding the patient by a tube passed through the nose or by enemata, and in trying to cut short the attack by faradisation with a wire brush or by the cold douche.

HYPOCHONDRIASIS AND NERVOUS EXHAUSTION.

Like hysteria, both of these are functional diseases, and are not attended with any known organic change in the nervous system. Hypochondriasis is a condition of mental depression, apparently without a sufficient cause, associated with the conviction in the patient's mind that he has some serious bodily disease; while nervous exhaustion, or neurasthenia as it is also called, is a condition of impaired nutrition of the nervous system, often associated with mental depression, and rendering the person unfit to perform prolonged physical or mental work.

Causes.—*Heredity* plays a distinct part in both diseases, and some nervous disease or insanity often exists in the father and mother or in other members of the family.

Age.—Hypochondriasis is a disease of middle life; neurasthenia affects any age from puberty upwards.

Sex.—Men are most affected with hypochondriasis, and women with neurasthenia; but men frequently have the latter.

The *determining cause* is most important. Hypochondriasis may be brought on by frequent slight ill health and by family worries, but it also attacks retired professional men who lead indolent aimless selfish lives; while neurasthenia is often the result of prolonged ill health or systematic overwork, attended with anxiety, especially working against time. Hypochondriacs have too much leisure time; neurasthenics too little or none at all.

Injuries such as severe accidents are exciting causes of neurasthenia.

Symptoms.—In *hypochondriasis* the main point is that the patient concentrates his attention on one particular part of the body, and believes that it is the seat of disease. He is always examining himself, and notes the slightest fur on his tongue, or alteration of his fæces from what he considers to be healthy. The condition is often started by slight dyspepsia, with some pain at the epigastrium, furred tongue, constipation, and flatulence, and this gives the patient the idea that he has cancer of the stomach; or from the effects of slight ill health and worry he gets frequent headaches, and is thereby convinced that he has a tumour of the brain. Various curious subjective sensations, such as that of the skull seeming to open and shut, or that of the brain feeling to move inside the skull, are complained of—symptoms which are not usually met with in tumours of the brain. Sharp burning pains occur about the limbs or tender points over the vertebral spines. In other cases there is severe uneasiness about the heart, lungs, and liver, and patients say that they can feel the food going down the œsophagus and stopping at a certain point. Hypochondriacs read up their case in medical books and worry their medical friends, and would like to monopolise the entire time of their doctors with endless questions, which they frequently have written down in a notebook.

In *neurasthenia* the typical symptom is an inaptitude to perform any work, whether physical or mental, and especially the latter.

Women after very hard, anxious work, such as nursing a relative, or even after following employment in an office, gradually find that there is a difficulty in concentrating the mind on business, while at the same time there is general languor, so that the patient can only walk a mile or so. Men are also liable to this condition, who continually work during prolonged hours of business, attended with much anxiety, and without taking any regular exercise or mental relaxation; or the symptoms appear soon after a severe accident.

The symptoms met with are headache, especially over the occipital region, and extending over the vertex to the root of the nose, as though the head was pressed upon or gripped in a vice, or as if the skull was opening ; pain and extreme tenderness over the vertebral spines ; numbness and tingling in the limbs without actual anæsthesia. There is no definite paralysis, but the movements of the limbs may be tremulous, and the knee jerks increased, and in testing them the patient often complains of severe pains shooting from the patellar tendon up to the spine—a symptom not met with in organic disease. Though there is no paralysis and the grasp is good, the patient complains of great effort in muscular movements and of easily getting tired. Other symptoms are palpitation and dyspepsia, with a furred tongue and constipation, or an inability to take food, so that extreme wasting may ensue.

On the mental side the patient is utterly unable to attend to business, any attempt bringing on headache, and confusion of thoughts with loss of the power of concentration ; there is much depression of spirits with a tendency to crying ; sleep is very bad, or attended with horrible dreams.

With all these subjective symptoms there are none which are objective, the whole condition seeming to depend on the depressed condition of the nervous system.

Diagnosis.—It is absolutely essential that in making a diagnosis of both these diseases, all the organs of the body should be examined, and especially the urine should be tested and the optic discs examined.

Although some neurasthenics become hypochondriacs, the two diseases differ in that the latter have a sort of monomania about some particular organ and the importance of their cases above all others, while there is not that severe depression of mind or body found in the neurasthenic cases.

Prognosis in hypochondriasis is better as the age of the patient is younger, the duration short, and in the absence of hereditary insanity; while in neurasthenia the prognosis is favourable if the cause can be removed, but there is a great liability to relapse if the circumstances are reproduced.

Treatment.—In both classes of cases it is most important to gain the patient's confidence, and this can only be done by examining every organ, and thoroughly going into each case. By this means alone can organic disease be excluded and the patient assured of the fact; but on no account should the patient's symptoms be laughed at or made light of. Those hypochondriacs who have no occupation should be told that they must take up a hobby, or some philanthropic work, etc., and should be prohibited reading medical works or their doctor's prescriptions, and should be told not to worry about the precise amount and colour of their excreta. The dyspepsia from which they often suffer should be treated by regulating the diet, and by dilute mineral acids and compound tincture of gentian, and flatulence by creasote or valerian. Regular exercise every day is most necessary, but should fall short of tiring the patient.

For the true neurasthenic case there is nothing which gives such good results as Weir-Mitchell treatment (see p. 375). This is especially the case with patients who have lost flesh and are unable to take food; but it is also very useful for even robust patients whose appetites are fairly good, but who are always tired and are unable to concentrate their minds. Great experience and tact are required to get these patients to undergo the treatment, especially during the first week or ten days. The treatment acts by giving the patients absolute rest, and by putting their minds into a different groove. After a month or six weeks of seclusion, a sea voyage or a stay at the seaside is of great use.

If the patient will not undergo the above treatment, all work must be absolutely prohibited, and rest for mind and body must be sought for in a sea voyage or mountain air; but it often fails if not preceded by the Weir-Mitchell treatment.

There are some cases of neurasthenia where Weir-Mitchell treatment fails, and those are the patients who are despondent and melancholic or who are very depressed, and it is of no use trying the treatment unless the patients are anxious to get well and will loyally co-operate in carrying out all the details of the treatment.

For want of sleep bromides are best with tinct. cannabis Indicæ; sulphonal may be given, but not chloral if it can be avoided.

TRAUMATIC NEUROSIS.

This, as the term denotes, is a derangement of the nervous system, occurring after an accident, which may be a blow on the head or on the back by a heavy weight falling on it, or a railway accident where the patient is thrown backwards and forwards in the carriage and severely shaken without any bones being broken.

Causes.—There is probably in most if not all cases a predisposing nervous temperament and an unstable nervous system; and there may be a family history of nervous diseases.

Age.—No age is exempt, and I have had a case as young as ten years and as old as over sixty.

Sex.—Both sexes are affected, and in my experience the male as often as the female. Both of the above-mentioned cases were males.

Symptoms.—In some cases the symptoms come on at once, while in others they are delayed for two or three days, so that at the time of the accident the patient can assist his fellow-sufferers; but after the excitement has passed over the patient in two or three days' time may gradually present the symptoms of neurasthenia. In other cases the symptoms are undistinguishable from those described on p. 367; there is anæsthesia and analgesia of the limb which was struck, beginning usually a short interval after the traumatism, and either involving that limb only, or extending into complete hemianæsthesia of that side with crossed amblyopia and achromatopsia of the eye of the same side, while there is some loss of power of the hand and foot with or without contracture. Tremor also occurs, and though it is much increased by voluntary effort it often persists while the patient is lying at rest.

The symptoms are probably due to the nervous system, and especially the highest cerebral centres, being depressed by the physical shock or fear of impending death. It has been considered that traumatic neurosis is a distinct entity, something quite apart by itself; but there does not seem to be any evidence for this, for though some of the above symptoms occur idiopathically, others like neurasthenia are brought on in some people by overwork and worry, and there is no reason why physical as well as mental shock should not be equally potent.

Diagnosis has to be made from malingering on the one side, and disseminated sclerosis on the other.

Prognosis is favourable if the cases are treated early; but it must be remembered that patients, who have once suffered from this complaint, are more ready than other people to be affected by subsequent worries and physical accidents, and that they are liable to a relapse.

Treatment.—The Weir-Mitchell treatment already described gives the best results in these cases; but it is of no use trying to carry it out at the patient's home, as is sometimes done; they must go away from their accustomed surroundings before any good can be expected. In a general ward of a hospital these cases do very well under Weir-Mitchell treatment, if put into a corner bed with screens kept continually round it to cut the patient off from the rest of the ward.

Internal treatment is not as a rule required, and it is very essential to avoid giving hypnotics like chloral; and if they have been already given, it is surprising how soon under the above treatment they can be dispensed with.

On recovery after the above treatment patients should be warned not to over-fatigue themselves mentally or physically, and they should be told to be careful in this respect for a year or two after, as it is probable that they will not be able to perform at first as much as they used to do. Cases which are not taken in hand soon are very liable to drift into chronic invalids.

CHAPTER XXVIII.

NERVOUS DISEASES FROM ALCOHOL AND METALLIC POISONS.

ALCOHOLISM.

THE effects of alcohol on the peripheral nerves have been already described (p. 89), and it will be only necessary here to describe the poisonous action on the brain, the symptoms of which may be described as acute and chronic.

ACUTE ALCOHOLISM (DELIRIUM TREMENS).

Causes.—*Heredity.* A tendency to excessive drinking is observed in other members of the family in about half the cases, while insanity or other diseases of the nervous system are met with, though to a less degree.

Age and sex.—It is unusual before twenty-five or thirty years old, and it affects men much more than women. *Occupations* as barmen, publicans, and commercial travellers are especially predisposing.

Injuries as a broken leg often bring on an attack, as well as acute diseases like pneumonia.

As regards the direct cause, alcohol in the form of spirits is more potent than as wine or beer, due probably to the less concentrated form of the latter; the attack may come on after a few days' heavy drinking, or less frequently after a steady excess of liquor, lasting for some time.

Symptoms come on rather rapidly, becoming fully developed in three or four days. The patient is at first restless, irritable, and he cannot sleep, the tongue is furred, there is constipation, and no appetite; there is tremor of the tongue when protruded, of the face muscles, and in the hands when held out in front; at first it is usually fine in character, and is increased by voluntary effort, but it subsequently becomes coarser.

At the onset the pulse is full, and the temperature is raised to 101° or more, and the patient perspires much. At night-time characteristic horrible dreams disturb the patient and make him very restless; he next has the dreams by day, with hallucinations of sight, which at first he can shake off; he then gets complete insomnia, followed the next day by persistence of the hallucinations, and he passes into the second stage, that of delirium, which may be of a maniacal form, so that he may attack his attendants or injure himself. The hallucinations take the auditory form of voices jeering at him, or the visual form, as demons or animals in the room, or horrid insects crawling over him.

The patient talks incessantly and incoherently. The tremors increase and affect all parts, and he is constantly picking at the bedclothes or trying to get up. The temperature is raised sometimes to 105°, or even higher, the pulse gets quicker and gradually weaker. The urine becomes diminished in quantity with high sp. gr., and may contain albumen. The pupils react to light.

The above symptoms continue for three or four days, the patient gradually getting weaker without any sleep, when he passes into a sound sleep lasting all day, and wakes up almost quite well. On the other hand, in unfavourable cases he gradually gets weaker, and dies in a comatose condition with epileptic convulsions. In other cases the profound sleep of recovery may not come till the end of a week.

Most cases of a first attack recover with only a slight tremor and weakness, which soon passes off; but second and third attacks are much more dangerous, and death may occur from exhaustion or syncope, or from the disease which has been the exciting cause of the delirium. Cases which recover after several attacks do so slowly and imperfectly, and some may end in dementia. A high temperature and a weak, soft, rapid pulse are bad signs, also the presence of traumatic shock.

Pathology.—Congestion of the brain has been described; but it is probable that the alcohol acts as a direct poison on the cortical cells in the same way as absinthe will produce epileptic fits, by its action on the cortical cells.

Diagnosis.—The history of heavy drinking and the characteristic ethereal smell of the breath, or a history of anorexia and morning vomiting for some time, will help the diagnosis; while the tremors, the insomnia, with horrible dreams passing on to hallucinations, and the extreme terror, will separate this affection from other mental diseases.

Treatment.—This consists, in controlling the patient so that he does not hurt himself or others, in the administration of food, and in procuring sleep.

For controlling the patient he must be watched night and day, and never left a moment alone; and if he be violent, he must be restrained by a "strait waistcoat," or be tied down with bandages or leather gauntlets and anklets to the bed, with a broad sheet going over the body and tied under the bed, or be put into a padded room.

If the patient will take it, solid food should be given in full quantities; or if it be refused, liquid food should be administered by force, a soft indiarubber tube being passed into the stomach through the hole in the gag used for the stomach pump, and strong soups and eggs beaten up poured through a funnel attached to the other end of the tube, or the patient may be fed by nutrient enemata.

Some recommend an emetic and purge at the onset, but owing to the depressing character of the malady, it is better to give only the latter in the form of gr. v of pil. hydrarg., followed by a saline aperient, as a drachm of mag. sulph. and 10 grains of mag. carb., when the patient is young and strong.

If there is great intolerance of food with vomiting, bicarbonate of soda 20 grains or 10 grains of carbonate of bismuth suspended in mucilage with 5 minims of acid hydrocyan. dil. may be given three times a day. As it is essential that sleep should be procured, chloral hydrate gr. xv, pot. brom. gr. xx, should be given, with tinct. cannabis Indicæ ♏xv or tinct. hyoscyami ʒss. to ʒj, every three hours till sleep is obtained. Paraldehyde ʒij to ʒvj in syr. aurantii, chloralamide gr. xxx, or sulphonal gr. xx to xl in single doses are useful. If all these fail, gr. $\frac{1}{10}$ to $\frac{1}{4}$ of a grain of morphia may be given hypodermically; but it is not advisable to give a second dose of morphia early in the disease, say until the third day, as there is a possibility of seriously injuring the patient and paralysing the heart without procuring sleep (Anstie). Extr. cannabis Indicæ in one dose of gr. $\frac{1}{4}$ to gr. $\frac{1}{2}$ is often efficacious. As a stimulant alcohol had better be avoided during the attack, and if there is much prostration ammonia or digitalis given instead.

Chronic Alcoholism.

This affects the nervous system chiefly in the form of tremors. The patient, who often has a bloated, flabby appearance about the face and watery eyes, complains of morning vomiting with absolute loathing of food, and a sinking at the epigastrium which is relieved by alcohol; he has a foul taste, and the tongue is covered with fur; there is the characteristic "oinosmia," or vinous odour in the breath, and on putting out the tongue and holding out the arms with the fingers extended and separated a fine tremor is noticed, which is increased by voluntary effort. The patient suffers from insomnia, and is irritable; he has buzzing in the ears and specks before the eyes, with loss of memory; the condition resembles general paralysis of the insane, but differs from it in that the pupils are equal and react to light and the knee jerks are not altered (unless there is neuritis); some cases, if not stopped in time, may go on to chronic dementia. The absolute untruthfulness of many alcoholics is proverbial.

Treatment of chronic alcoholism is to enjoin absolute abstinence from all alcohol, and many hospital patients are not aware that the alcohol is the cause of their troubles, and frequently will give it up when the matter is explained to them; the presence of oinosmia a week after alcohol has been stopped will show at once that the patient has not observed abstinence, and he should be told so at once. Bicarbonate of sod. gr. xx with tinct. gentian. co. ʒss. and tinct. capsici ♏iij to x three times a day before meals, or quinine in 1-grain doses three times a day with bicarbonate of potash and citric acid (Anstie), should be given to improve the gastric conditions; while if there is insomnia, 40 grains or a drachm of potassic bromide or from $\frac{1}{4}$ to $\frac{1}{2}$ grain of extract of cannabis Indica at night-time. As regards other symptoms, cod-liver oil and liquor strychninæ in 2-minim doses are useful in advanced cases where there is much weakness and tremor. In repeated cases the patient should be strongly urged to put himself voluntarily under restraint.

LEAD PARALYSIS (SATURNISM).

Lead, when taken into the system in repeated small doses, affects the nervous system in the form of loss of power and of sensation in the limbs, accompanied by pain, or less frequently there is general cerebral disturbance, with convulsions, passing on to coma.

Etiology.—Although no hereditary tendency has been determined, some persons seem to have a special proclivity to be affected by lead. No age is exempt, and women are more liable than men. The disease, which predisposes to lead poisoning, is gout. The detection of lead as a cause is not difficult in cases of plumbers, painters, and other trades who use lead or the carbonate of lead; but in people who are not in apparent contact with lead it is often difficult. In the former case it is usually taken into the mouth by the men neglecting to wash their hands before meals, and in the latter through drinking water kept in leaden pipes or cisterns, from snuff which had been wrapped in lead paper, from cider or acid stewed fruits kept in porcelain vessels glazed with lead, or from using lotions and washes containing lead. The lead may thus be absorbed by the alimentary canal or by the skin, and in animals which have been fed on lead it has been found after death in the spinal cord and brain, and in the muscles, as well as in other organs.

Symptoms.—The chief symptoms which affect the nervous system are paralysis of certain muscles, and in other cases cerebral disturbance with mental changes.

Paralysis occurs in two forms, acute and chronic.

The acute form is the one most commonly met with, and it presents the typical wrist-drop of lead paralysis, and affects the two sides symmetrically. The first symptom is usually weakness in extending one finger of one hand, usually the right; and this spreads to the other fingers, and then to the thumb, the extensor ossis metacarpi being often affected last. There may be no pain or slight sharp pains along the nerve trunks of the limb, with some tenderness of muscles; while in other cases there are painful spasm or cramps in the muscles. The difficulty is especially in extending the metacarpo-phalangeal joints. Later on the extensors of the wrist become affected, and then the wrist is completely "dropped," and cannot be moved into a line with the forearm. In examining movements of the wrist, care must be taken to fix the forearm, and this is best done by laying the limb on a table so that the hand hangs over the edge, and then noticing if the hand can be raised at all out of its hanging position. All the muscles on the back of the forearm are affected except the supinator longus, which is usually conspicuous by escaping, although it is supplied from the same nerve, the musculo-spiral, and it can readily be made to contract by the patient flexing his elbow against resistance. The flexors of the fingers are not affected, and their apparent weakness is due to their acting at a disadvantage, their muscles being shortened owing to the wrist not being fixed by the paralysed extensors of the carpus; but if the wrist be kept passively extended, the flexors of the fingers can act powerfully. Later on the muscles of the upper part of the arm may be involved, and especially the biceps, brachialis anticus, and deltoid, and in this case the supinator longus is also affected. In the lower limbs the muscles on the front of the leg may be affected, but the tibialis anticus may not be involved.

General tremors with hyper-excitability to percussion are sometimes observed with twitching of the muscles, resembling general paralysis. The muscles rapidly waste, and show changes to electrical testing; to the faradic current excitability is diminished or completely lost, while to the galvanic current excitability is increased, and the contraction to the positive pole is obtained with a weaker current than with the negative (see p. 73). Erb has found this alteration in muscles before they are paralysed. The muscles may recover power before the faradic excitability returns.

Sensation is not affected, or there may be a patch of slight tactile anæsthesia or analgesia over the part supplied by the affected nerve.

In the chronic form, which is less common than the acute, the symptoms resemble those of progressive muscular atrophy (p. 158). There is a slow, progressive atrophy of the small muscles of the hand with fibrillar tremors, and with loss of power in proportion to the wasting, and to electric testing the muscle and nerve give the partial reaction of degeneration (p. 73)—*i.e.* the nerves require stronger faradic and galvanic currents to react, and the muscles require a stronger faradic current, but with the galvanic they react more readily but slowly, and better to the positive than to the negative pole.

Some cases have been described (Philipson) where all the limbs and the trunk muscles have been paralysed on successive days, so that only the muscles of the abdomen, chest, neck, face, and eyes were unaffected. In other cases the ocular muscles have been attacked (Buzzard). Cerebral symptoms occur sometimes early in lead poisoning, as convulsions, followed by delirium and coma, and preceded by giddiness, noises in the head, temporary blindness, and general restlessness. The convulsions are epileptic in nature, and are either general or affect one side the more.

It is in the early stages of these cerebral cases that optic neuritis occurs with swelling of the disc and hæmorrhages, passing on sometimes to atrophy with loss of vision; or changes in the disc may occur, as albuminuric retinitis, in the later stages associated with renal disease (Oliver). Melancholia with delusions may be met with, and sometimes the cases much resemble general paralysis.

Besides the above symptoms, others characteristic of lead poisoning must be looked for, such as the white anæmic face, and the general wasting of the body and loss of strength. The blue line which is observed along the edge of the gum, especially in the lower jaw and where the teeth are dirty and there is much tartar, is most characteristic, and it appears as a fine line, and when viewed with a lens this is seen to be made up of small dots; it is a deposit of sulphide of lead in the vessels of the gums, or an absorption from the mucous surface either by the lymphatics or by the cells themselves (Oliver).

Attacks of colic, with constipation, retraction of the abdomen, and attended sometimes with vomiting, are frequent symptoms.

The general symptoms of lead poisoning, the pallor, general wasting, and cachexia with occasional colic, may exist for several months or a year or two before paralysis occurs.

The kidneys are liable to be affected with interstitial, passing on to granular, nephritis.

Pathology.—In the nervous system the seat of lesion differs according to whether the symptoms are acute or chronic.

In the common acute form of double wrist drop, the changes are essentially those of a peripheral neuritis, having a special selection for the nerves supplying the extensors of the fingers and wrist. The motor or the intra-muscular nerves suffer most, as anæsthesia is not common. The changes in the nerves consist of a cloudy granular change in the medullary sheath, which becomes segmented and broken up; later the axis cylinder breaks up till the nerve is quite disorganised. The disease is more marked nearer the periphery than towards the cord, and this has been given as a reason why the extensors of the wrist and fingers are most affected; but this cannot be so, as the interossei and small muscles of the thumb should be first affected. It is more probable that these muscles are affected because they and the extensor of the toes are in a subordinate position, for they do no work beyond putting the fingers and toes in a position to be able to begin working. The muscles are wasted, and the fibres are segmented and lose their striation and in time disappear, leaving fat and connective tissue. These changes have been by some considered to be primary, but they are more likely secondary.

The spinal cord is not as a rule affected, except in the slow chronic form of progressive muscular atrophy, when atrophy of the cells in the anterior cornu has been observed.

No definite changes have been found in the brain, except dryness and contraction of the vessels, and probably during life there is increased intra-cranial pressure (Oliver).

Diagnosis has to be made by the appearance of the double wrist drop, the pale cachectic appearance, the blue line on the gums, and the attacks of colic. From acute or subacute polio-myelitis the diagnosis is made, in the absence of the blue line and colic, by the improbability of the disease being so symmetrical and so limited as in lead paralyses. In alcoholic multiple neuritis there is much more tenderness of the muscles, and the extensors of the toes are more commonly involved, while there is a history of alcohol and not of lead. In paralysis of the muscular spiral nerve the affection is usually unilateral, and the supinator longus is then involved early. The chronic progressive form may be impossible to diagnose from ordinary progressive muscular atrophy without other signs of lead.

Prognosis.—If the patient can be kept from lead contamination, recovery from wrist drop is usual, though it may take some time—*i.e.* from two to twelve months. The unfavourable conditions which may cause death are cerebral symptoms and the presence of renal disease with arterial degeneration. Recovery from the chronic form frequently does not occur, the condition remaining stationary.

Treatment consists in removing the patient from the influence at once, and he should not be allowed to work with lead for three months. Preventive treatment is very important, especially washing the hands and the nails before handling food. At lead works a "lemonade" containing dilute sulphuric acid is freely given to the workers, and respirators used where there is much dust.

The remedies which are of the most use are sulphate of magnesia and iodide of potassium; but the latter should not be given if acute symptoms are present, as a soluble lead salt is formed which may increase these symptoms before the lead is eliminated by the kidneys. It is best to begin with sulphate of magnesia with acid sulph. dil., to which some tinct. opii may be added if there is much colic, or an enema may be necessary if the constipation is obstinate, and then to give iodide of potassium two or three days later. For the paralysis strychnine and nux vomica have been used, but beyond a general tonic their specific action on the muscles is doubtful.

The systematic treatment by electricity, using the constant current, is useful, the positive pole being applied to the spine and the negative brushed over the muscles, using a current just strong enough to make the muscles contract.

In the acute cerebral symptoms amyl nitrite is recommended, and for the fits, especially if accompanied by suppression of urine, hypodermic injections of pilocarpine (Oliver). The bromides and chloral given as an enema may be required if the fits are frequent.

ARSENICAL POISONING.

Poisoning by small doses of arsenic is occasionally produced either by taking the drug medicinally, or more frequently from cosmetics or sleeping in rooms with arsenical wall papers.

Symptoms are acute, and consist of numbness and tingling, and later of sharp burning pains in the limbs, with tenderness along the nerve trunks and hyperæsthesia of the muscles, followed by anæsthesia of the skin and loss of power in the extensors of the wrist and digits and also of the toes. The muscles rapidly waste, and give the reaction of degeneration with electrical testing, and if the extensors of the knee are involved the knee jerk is lost. I have seen permanent paralysis of the extensors of the hands and feet occur after taking ♏xv of liq. arsen. ter die for six weeks.

Less frequently acute symptoms resembling the ataxia of tabes dorsalis are observed, and associated with severe pain and atrophy of muscles.

A curious discoloration of the skin occurs in the form of brown pigmented spots, and this is seen after taking arsenic in small doses for some time, and is unattended by any of the above symptoms (Hutchinson). In other cases changes occur in the hair and nails.

Diagnosis has to be made from paralysis due to lead and to alcohol, both of which arsenical poisoning resembles. The absence of a blue line and of any history of poisoning by these two, and the symptoms of ataxia associated with tenderness of the nerve trunks, should help the diagnosis. The paralysis due to arsenic has less tendency to recover than after lead or alcohol.

Treatment consists in stopping the source of the poison, giving iodide of potassium, and treating the muscles by the constant current.

MERCURIAL POISONING.

Poisoning from this metal is not very often met with in this country, and when seen it is the result of handling mercury, as in looking-glass makers, and not from being taken as medicine.

Symptoms.—The nervous symptoms are often preceded by ulceration of the mouth and gums, with salivation and fœtid breath. The most characteristic symptom is a very fine tremor affecting the face, tongue, then the arms, and lastly the legs; at first only on voluntary movement, but soon becoming persistent, though it is always increased by voluntary effort. The speech becomes difficult and stammering. The muscular power is diminished, but never completely lost, and there are no electrical changes and no wasting of special muscles. Numbness, tingling, pains in the limbs are present, and sometimes slight analgesia.

Mental symptoms, as irritability and restlessness, going on to mania, have been observed.

Diagnosis.—The diagnosis in the absence of a history of mercurial infection or of stomatitis has to be made from disseminated sclerosis in the early stage and from paralysis agitans or general paralysis of the insane in the later; but the absence of all other symptoms of these diseases besides tremor should cause no difficulty.

Treatment.—Most cases recover when removed from the influence of the metal, while iodide of potassium in small doses is considered to help to remove the poison.

CHAPTER XXIX.

PARALYSIS AFTER ACUTE DISEASES, OR DUE TO A SPECIFIC VIRUS.

SYPHILITIC DISEASES OF THE NERVOUS SYSTEM.

Syphilis enters so largely into the causation of diseases of the nervous system, and it has been referred to in so many parts of this work, that it will only be necessary to summarise the chief conditions met with.

In the peripheral nerves of a limb, or in the cranial nerves, a neuritis may occur, accompanied by a cellular growth within the sheath of the nerve similar to syphilitic growths elsewhere.

In the spinal cord the specific process affects, (1) the meninges, producing a thick, gelatinous condition of the membranes, with infiltration of small round cells in the pia mater, and spreading to the periphery of the cord; (2) the arteries, producing blocking of the vessels with consequent softening of the parts of the cord supplied by them; (3) causes distinct gummata of the cord and meninges, usually multiple. (1) Specific meningitis affects the cervical region, with severe pain in the spine shooting down the arms and worse at night, with a rigid spine, tender to percussion, but without pyrexia; this is followed by hyperæsthesia and anæsthesia of the arms, and wasting of a group of muscles corresponding to the distribution of the involved roots. If the meningitis spreads to the cord (meningo-myelitis), both legs have tingling and numbness with paralysis of movement, and affection of the sphincters, later with increased deep reflexes and rigidity. (2) Specific arteritis produces rapid acute loss of motion and sensation with retention of urine, in a few days or sometimes hours, followed by bed sores, and frequently a fatal termination (see "Acute myelitis," p. 121).

Besides the changes in the cord due to direct syphilitic lesions, there are the degenerations found in locomotor ataxia which are indirectly due to the syphilitic poison.

Professor Erb describes a gradual paralysis which he considers specific, beginning with numbness and slight pains in the legs, vesical troubles, spastic condition of legs in walking, but less on sitting down, with increased knee jerks and ankle clonus, but without wasting or loss of power, and considered by him amenable to anti-syphilitic treatment. I have seen such cases, but I cannot say that they always respond to the treatment.

In the brain, as in the cord, syphilis may affect the meninges, the brain substance, or the blood vessels.

The pia-arachnoid membranes are affected (1) by a thick gummatous exudation almost like gelatine, especially at the base of the brain involving the cranial nerves, causing first irritation and then paralysis, and rarely all the membranes are involved; this exudation may become organised into thick fibrous bands, as in a case of mine where the cranial nerves were involved in fibrous bands of the pia mater, producing blindness of the left eye, right hemianopia of the right eye with hemiopic pupil (p. 195), paralysis of the right third nerve and the left seventh, and temporary paralysis of one fifth nerve; (2) by gummata growing from the pia mater, or sometimes from the dura mater, and extending along the vessels into the brain substance; they are found in the frontal and parietal regions.

The brain substance is the seat of gummata, which have grown in from the surface, and they are found in the cerebral peduncles and pons Varolii, rarely in the basal ganglia and cerebellum. In some cases a diffuse gummatous infiltration occurs in the cortex, while in others circumscribed gummatous patches are found on the surface of the pons and peduncles.

The large, cerebral arteries are particularly affected by a syphilitic arteritis, which produces a cellular growth between the inner coat and the elastic lamina, causing a projection into, and a narrowing of, the lumen of the vessel. Here a thrombus is very liable to form, leading to softening of the part of the brain supplied by the vessel; or if the cellular growth becomes fibrous, an aneurysm may arise, and by giving way may produce a hæmorrhage.

AFTER DIPHTHERIA.

Paralysis after diphtheria has been long known, and is probably the most frequent example of paralysis following an acute disease.

Predisposing causes.—*Age.* Young adults are most commonly affected, though no age is exempt, and there is no difference between the sexes.

The previous health of the patient and the severity of the throat attack have no connection with the paralysis, which may occur after a sore throat which was so slight as hardly to be noticed; and probably all sore throats followed by this form of paralysis are diphtheritic.

About a quarter of those who survive the diphtheritic throat attack suffer from paralysis (Gowers).

Symptoms consist of a gradual paralysis coming on without pain, and as a rule without pyrexia, one or two weeks after the throat has recovered, and affecting first the soft palate or the mechanism for the accommodation of the eyes. The patient cannot read, or there is difficulty in swallowing, and fluids regurgitate through the posterior nares. On trying to blow up the cheeks, this is found to be impossible, owing to the air escaping through the nose; from the nasal cavity not being shut off, explosive sounds as p, b, d, t cannot be pronounced, so that "rub" becomes "rum"; if the anterior nares are closed by pinching the nose, the power of inflating the cheeks returns, but not the natural speech. On examining the throat the soft palate hangs motionless, and is not drawn up on phonation or on a deep inspiration.

After the throat and ocular paralyses, the limbs may become affected with tingling and numbness, and then with weakness, but unusually with complete paralysis, the legs being involved before the arms. The weakness is bilateral, and the knee jerks are lost early, even when there is no appreciable loss of power (Bernhardt). In slight cases the patients drag their legs in walking, and feel generally weak, while the grasp of the hands is deficient, and there is difficulty in raising the arms; there is an absence of all rigidity, but in severe cases the limbs may be almost completely paralysed and flaccid. The characteristic course is for one part to be attacked while another part is recovering.

At first there is no change to electric stimulation, but later on the nerves lose their irritability and require strong faradic and galvanic currents to react, and subsequently they will not react at all, and the paralysed muscles give the reaction of degeneration (p. 73).

The ocular muscles are sometimes paralysed, especially the internal recti, but the pupils react to light. In severe cases all the muscles of the body are involved, the trunk and back muscles, the neck muscles, the intercostals, the larynx, pharynx, and even the diaphragm; but owing to the successive manner in which the parts are attacked, the two latter are usually not involved at the same time. Cardiac paralysis, as shown by irregularity or by slow or rapid action, may occur.

Affection of the sphincters with incontinence or retention is very uncommon, even when most of the voluntary muscles are paralysed.

When the limbs are involved, the patient complains of tingling and numbness, which may pass on to actual anæsthesia and analgesia when the paralysis is well marked; but the distribution is very irregular, and does not correspond to that of any peripheral nerve or spinal root. There is not as a rule tenderness over the nerve trunks or of the muscles similar to that of alcoholic neuritis.

The duration is very variable, being prolonged by the successive attacks of different parts of the body, and it may last one or two months or longer, according as the palate and the ocular muscles of accommodation are alone affected, or the limbs as well. A part once paralysed is not involved again in the same attack, and the more severely a part is paralysed, as shown by the loss of faradic excitability, the longer will it be affected, and more likely to be followed by paralysis elsewhere.

Prognosis.—The prognosis for ultimate complete recovery is good, unless the patient is much depressed by the preceding disease and cannot take food, or the heart or the muscles of respiration are affected; but in most cases the muscles gradually recover power, their electrical excitability returns later, and lastly the knee jerk, which may be absent long after the return of power in the extensor cruris, is obtained.

Pathology.—The changes found are distinctly those of a peripheral neuritis, and consist of a degeneration with segmentation and breaking up of the white medullary sheath, the axis cylinder persisting or being destroyed according to the severity of the case. The most important points are, firstly, the very wide distribution of the neuritis, affecting almost any nerve in the body, from the nerves of the palate, where the changes were first discovered (Charcot and Vulpian), to those of the limbs, or to the phrenic; and secondly, the limited area of the changes in the nerve, so that a patch of degeneration may involve the whole thickness of a nerve while the rest of it appears healthy.

In the spinal cord no changes have been found except a granular, vacuolated, or shrunken condition of the anterior cornual cells.

As to the cause of the changes in the nerves, they are probably due to a poison; this according to Martin is an albumose formed by a ferment which is itself produced by the diphtheritic bacilli, and certain motor nerves, especially those of the palate, are particularly susceptible to this poison.

Diagnosis.—After an attack of sore throat, difficulty of swallowing with nasal speech and regurgitation of fluids, paralysis of accommodation, and slow progressive weakness of the limbs with loss of the knee jerks, are almost certain to be symptoms of diphtheritic paralysis. The diagnosis has to be made from other forms of neuritis, as alcohol and lead, in which there is probably a history of these poisons, and where the soft palate is not affected; also there is not that tendency for one part to be attacked while another is getting well. Infantile paralysis is more rapid in its onset, and is unattended by loss of sensation; while diphtheritic ataxia is more rapid in its onset than true locomotor ataxia. From hysteria the paralysis of the palate and the absent knee jerks will readily differentiate.

Treatment.—As most cases tend to recover, provided that the patient can survive the effects of the disease, the most important treatment is to keep up the strength of the patient by careful feeding and giving stimulants if the pulse be weak. If the soft palate is paralysed, semi-fluid food of the consistency of thick gruel is more easily swallowed than fluids; but if this gives difficulty, it is necessary to feed by a soft india-rubber tube passed into the œsophagus through the mouth or nose, or by nutrient enemata. If the patient refuse food, it is absolutely essential that he should be fed by force by one of the above methods.

As the tendency of the paralysis is to recover, it is very difficult to say whether any medicine has power to cut short the disease; in most cases, however, it is best to give a tonic, as quinine and iron, to keep up the strength of the patient. Belladonna has been considered to be useful in large doses (Abercrombie).

Cardiac failure and respiratory failure require especial attention. For the former stimulants, with strychnine or digitalis, are required, and the patient should be kept absolutely quiet; while for paralysis of the diaphragm and intercostal muscles, which fortunately do not usually both occur at the same time, artificial respiration should be carried on systematically for fifteen minutes, three or four times a day. Dr. Pasteur has pointed out the frequency of collapse of the lung in these cases, and advises this treatment as soon as the first sign of diaphragmatic paralysis appears—*i.e.* overaction of the lower ribs in breathing.

As regards local treatment of the paralysed muscles, gentle rubbing and also electric treatment should be employed; faradisation should not be used, but a weak constant current, only just sufficient to cause contraction of the muscles.

AFTER ACUTE SPECIFIC DISEASES.

Paralysis may occur after other acute specific diseases, as typhoid fever, small pox, measles, in the form of spinal myelitis, either anterior polio-myelitis or disseminated myelitis; but of greater importance is the paralysis found after influenza.

The attack may come on during the acute stage of influenza, when it takes the form of cerebral or spinal meningitis, or myelitis, or may occur a week or two after the acute symptoms of influenza have passed off.

Of these sequelæ, pains are the most important symptoms, occurring in the same positions as in the acute disease, and having a neuralgic character ; while in other cases melancholia, sometimes with stupor or dementia, are met with, and seem to be a continuation of the extreme mental depression characteristic of influenza.

Hysteria and epilepsy have also been described as occurring soon after influenza.

Cases have also been observed having all the symptoms of peripheral multiple neuritis.

As there is nothing special about these diseases, their treatment is the same as that given under the different diseases ; there is, however, after influenza one important point to bear in mind—*viz.* to get the patient away to a change of air as soon as possible, and to use stimulants freely if necessary.

TETANUS.

This is an acute disease, affecting with a continuous tonic spasm, varied by frequent severe exacerbations, the muscles, especially of the face, jaws, and back, and produced by a definite organism. Several forms are described—the traumatic, the idiopathic, the puerperal, and tetanus neonatorum.

Etiology.—*Age.* It occurs in new-born infants, but no age is exempt. Men are much more often affected than women, and dark races more than whites. The disease also appears to be more frequent in the tropics.

The immediate *cause* is a wound by which the infecting organism is introduced. Exposure to wet and cold has been considered to be a cause; but whether this is really a coincidence has not yet been settled.

Symptoms.—The first sign occurs from a few days to two weeks after a wound, and consists in a stiffness and slight pain about the muscles of the jaw and neck; but in a few hours there is difficulty in opening the mouth, owing to spasm of the masseters, and some trouble in swallowing; the muscles in the neck become stiff, and especially the extensors. This spasm gradually travels down the back, involving the extensors of the spine. The face muscles are affected, especially the depressors and the retractors of the angles of the mouth, which are thereby drawn backwards and downwards, giving rise to a set grin or "risus sardonicus"; the limbs are soon affected with spasm, but the legs more than the arms.

In a day or two, when the disease is fully developed, the chief characteristic is the violent *tonic* spasms occurring in the muscles, which are already in a state of contraction; the risus sardonicus is accentuated, the teeth are firmly clenched, and the tongue may be bitten; the extensors of the neck and trunk and those of the lower limbs are powerfully contracted, giving rise to opisthotonos—*i.e.* arching of the spine concave backwards so that the patient may rest only on his head and heels. In addition the arms are adducted at the shoulder and extended at the elbow, the abdominal and thoracic muscles may be involved causing temporary stoppage of respiration in inspiration, and sometimes the diaphragm is affected with spasm. The passing of urine may also be prevented by the spasm, and the bowels are constipated.

Each attack lasts from five to ten seconds, and is frequently repeated according to the severity of the case. The spasm is attended with severe cramping pains, the patient sweats profusely, the pulse is small and increases in frequency during the spasms, the temperature is often normal, or may be gradually raised during the attack, and just before death hyperpyrexia may occur, and continue rising after death (Wunderlich). There is no loss of consciousness in the attacks, and they usually cease during sleep.

The cases last from a few days to two or three weeks, when, if they recover, the spasms get gradually less, passing again into the tonic condition, and as at the commencement the parts which were first affected are the last to lose the rigidity.

The number of cases which recover are very small, the idiopathic form being more favourable than the traumatic, puerperal, and that of new-born children. The mortality given by various authors in the traumatic form is about 90 per cent., and of the idiopathic 55 per cent., while puerperal tetanus is almost always fatal. The fatal termination is either by asphyxia from the prolonged spasm of the respiratory muscles, or from syncope or general exhaustion.

Pathology.—It has now been shown that the disease is due to an infective disease from a chemical poison, produced by a bacillus introduced into an abraded surface. The organism exists in the soil, and is most easily inoculated by lacerated wounds where earth is introduced. In puerperal cases and in new-born children the poison is introduced along the genital tract and at the umbilicus respectively. In idiopathic cases the bacillus probably gets access to the system, but how is not known. The bacillus consists of straight rods about $\frac{1}{8000}$ of an inch long with spores at one end, and they are unable to grow in oxygen; when grown in suitable media, the bacilli when separated by filtration are found to be inert, while the products of the bacilli produced tetanus when inoculated into other animals (Vaillard and Vincent).

The morbid changes in those who have died of tetanus are congestion of the different organs, with rupture of the muscles from the violent spasm, or extravasation into their fibres. No definite changes in the nervous system have been found, except a neuritis of the nerve which may have been involved in the wound. The spasms are probably due to action of the poison on the pons, medulla, and spinal cord.

Diagnosis.—This has to be made from the disease most resembling it, *viz.* hydrophobia; but in the latter the spasms affect the muscles of swallowing and attempts at drinking, and the trismus of the jaw is absent, while there is an intense feeling of suffocation.

From strychnine poisoning the diagnosis is more difficult, as probably the seat of the lesion is in the same part of the nervous system; but the spasms begin usually in the back, reflex action is very active, and a spasm is readily excited by a physical jar.

Treatment.—As the poison is probably produced at the wound, it should be freely treated by a strong antiseptic, as nitrate of silver from 1 to 2 grains to the ounce, and the part excised.

The general treatment of the patient consists in keeping him absolutely quiet in a dark room, and in giving strong nutritious food. If the trismus prevent food being introduced, he should be fed with a stomach tube three or four times a day, relaxing the spasm if necessary under chloroform.

To modify the spasm or to procure sleep the bromides and chloral in full doses of half to a drachm of each three or four times a day are required, and to be continued till the spasm ceases. Morphia may be employed hypodermically in gr. $\frac{1}{6}$ to $\frac{1}{2}$ to procure sleep if the above drugs fail. In very severe spasms the inhalation of chloroform or nitrite of amyl have cut short the attack, and artificial respiration should be practised if the breathing cease in a paroxysm.

Calabar bean, gr. $\frac{1}{8}$ of extr. physostigmatis, every hour, or sulphate or salicylate of eserine hypodermically, beginning with gr. $\frac{1}{60}$ and in increasing doses, have been found useful by some observers.

Recently cases have been subjected to the antitoxin treatment, and with very encouraging results, on the principle that the serum of an animal rendered insusceptible to a disease would, if injected into another animal, cure the disease (Tizzoni, Behring, Kitasato). For this purpose the serum of a horse, which has been made immune by repeated small doses of the filtered tetanus toxins, is injected hypodermically into a patient suffering from tetanus in amounts of from 20 to 40 cubic centimetres for the first dose, followed by from 10 to 20 c.c. every six to twelve hours after; and of the fifty cases recently collected the mortality by this treatment was only 32 per cent. (Hewlett), but this is probably too low a figure.

HYDROPHOBIA (RABIES).

Only a few words need be said about rabies, which, like tetanus, is an acute disease due to a specific virus, and never arises except by inoculation through the skin or mucous membranes. It is one of the most terrible and distressing of diseases.

Symptoms.—After a bite the patient may have pain in the healed wound, and a general sense of uneasiness and fear; this after some weeks passes on to increased restlessness, and then the characteristic spasms occur with hyperæsthesia of the skin, so that the slightest draught brings on a spasm, which affects first the muscles of swallowing. Any attempt to drink brings on a violent spasm of the pharyngeal muscles and an elevation of the larynx, as in swallowing; at the same time there is strong spasm of the muscles of inspiration, with an intense feeling of suffocation, although there is no obstruction to the entry of air. The temperature, pulse, and respiration are all increased in this condition.

Mental symptoms may occur with delusions and hallucinations, or the respiratory spasms may spread to the whole body, producing tonic convulsions, sometimes with opisthotonos.

At the end some cases pass into the paralytic condition observed in animals, beginning with weakness in the lower limbs and gradually ascending; and this so resembles "Landry's ascending paralysis" (*q.v.*) as to suggest that this latter may really be a form of rabies (Horsley).

Death occurs in the convulsive form from syncope and exhaustion, and in the paralytic form from coma.

Pathology.—According to M. Pasteur, the quickest time that symptoms occur in animals after inoculation is about from seven to fourteen days, while in man it may be from six weeks to as many months or more.

The poison, which has not been isolated, is found after death most in the nervous system, and in consequence the whole nervous system is found to be hyperæmic, with exudation of leucocytes into the perivascular lymphatics. The stomach is congested, and in animals often contains altered blood besides hay and other foreign bodies; the trachea and bronchi are congested, and the lungs show signs of acute broncho-pneumonia with hæmorrhages.

The incubation period is earlier in children, and in those who have bites about the face, head, and hands, while punctured wounds are the worst.

Diagnosis has to be made from tetanus, strychnia poisoning, and hysteria.

Treatment.—Immediately after a bite or scratch on a limb a ligature should be put round it, and the wound washed well and cauterised at once with the actual cautery or a stick of silver nitrate, and then the part should be excised. The only preventive treatment which is of any use is that of M. Pasteur—*viz.* the injection hypodermically of a certain quantity of an emulsion of the dried spinal cord of rabbits which have died from the acute form of hydrophobia; this treatment must be carried out for several successive days, as soon after the primary bite as possible. To prove that the dog had rabies, it is necessary to inject a rabbit with an emulsion of the dog's dried spinal cord, when the absence or presence of symptoms will determine the condition of the dog. By this treatment of M. Pasteur the mortality has been reduced from 15 to 1·3 per cent. (Horsley).

If the above treatment cannot be carried out and the attack comes on, the patient should be kept in a dark room absolutely quiet, nutritious food must be given, and if he cannot swallow he must be fed per rectum.

Sedatives, as hypodermic injections of morphia, or chloral and bromide in full doses by enema, have in some cases seemed useful.

DESCRIPTION OF PLATES.

PLATES I—V.

Diagrams of the Cutaneous Areas of the Peripheral Nerves.

Plate
I. Head and Neck (after Thain).
II. Upper Limb, front (Lewis Jones).
III. ,, ,, back ,, ,,
IV. Lower Limb, front ,, ,,
V. ,, ,, back ,, ,,

PLATES VI—XI.

Diagrams of the Areas of Sensation supplied by the Segments of the Spinal Cord at the Levels of the Different Roots, the Even Numbers of which are represented on the Right Side and the Uneven on the Left Side of Each Diagram, and by the Medulla (*Head*).

These areas represent the segmental distribution of the Sensory Fibres of the Spinal Cord.

They were mapped out as the results of observations—

(1) On the areas of superficial tenderness which accompany referred visceral pain.

(2) On the distribution of Herpes Zoster.

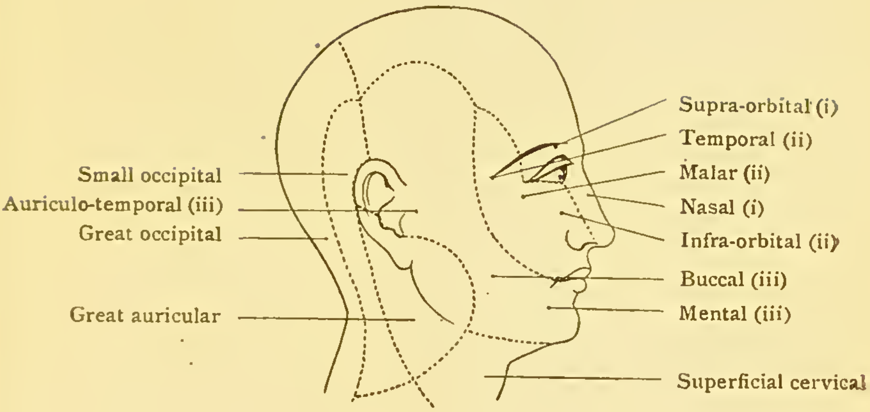
(3) On the distribution of the Anæsthesia in cases of transverse division of the Spinal Cord at different levels.

PLATES XII, XIII.

Diagrams of Loss of Sensation from Lesions of the Cervical Roots, and of the "so-called" Motor Cortex Cerebri, respectively.

*

PLATE I.



The points where the nerves become superficial are shown by the dots at the end of the pointing lines.

(i), (ii), (iii) denote that the branch belongs to the first, second, or third division of the fifth cranial nerve.

PLATE II.

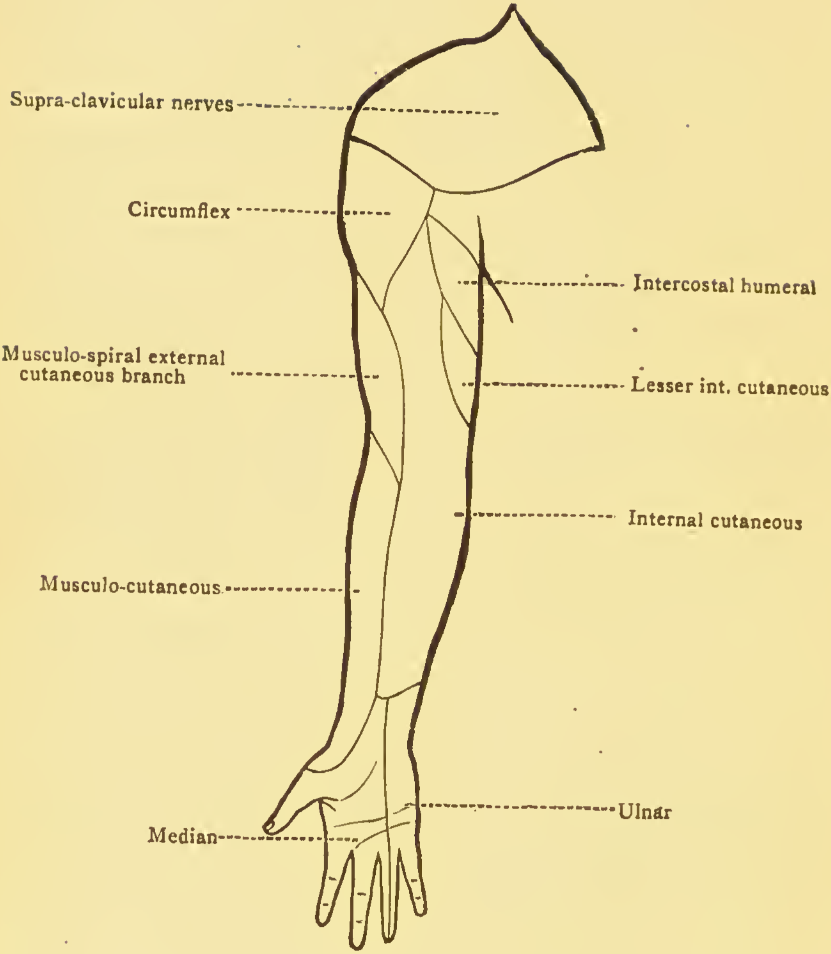

Supra-clavicular nerves
Circumflex
Intercostal humeral
Musculo-spiral external cutaneous branch
Lesser int. cutaneous
Internal cutaneous
Musculo-cutaneous
Ulnar
Median

PLATE III.

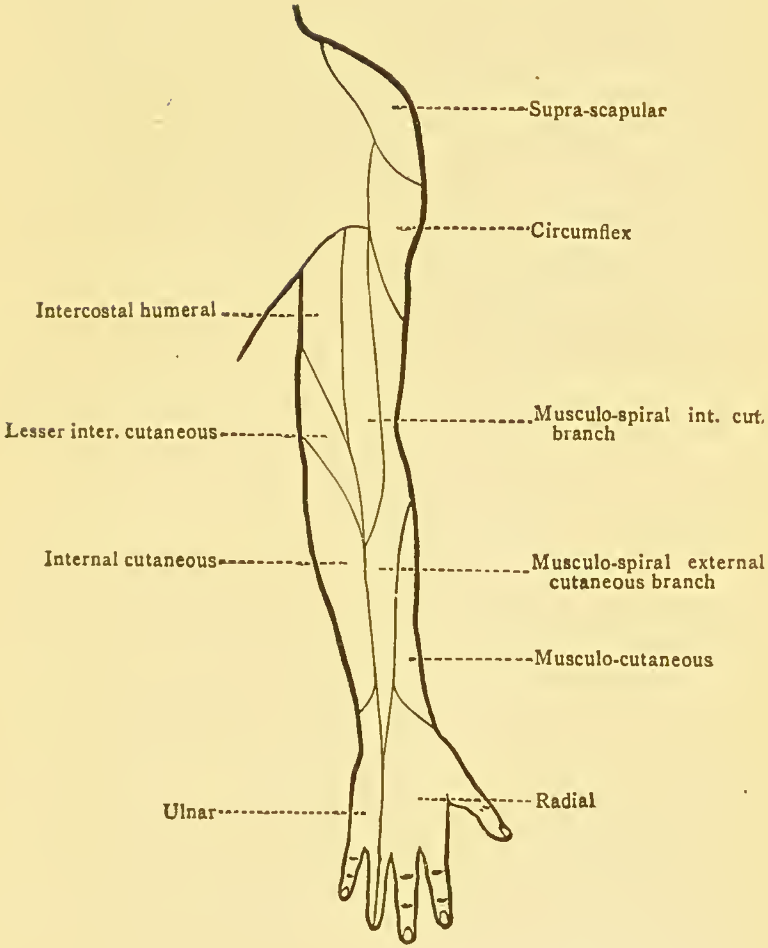

Supra-scapular
Circumflex
Intercostal humeral
Lesser inter. cutaneous
Musculo-spiral int. cut. branch
Internal cutaneous
Musculo-spiral external cutaneous branch
Musculo-cutaneous
Ulnar
Radial

PLATE IV.

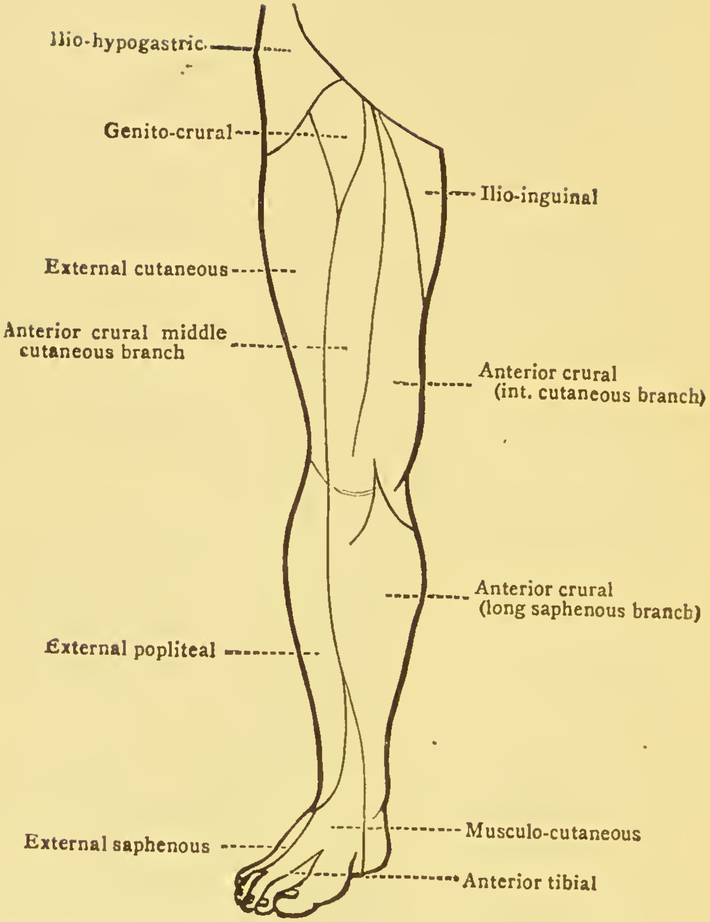

Ilio-hypogastric
Genito-crural
Ilio-inguinal
External cutaneous
Anterior crural middle cutaneous branch
Anterior crural (int. cutaneous branch)
Anterior crural (long saphenous branch)
External popliteal
Musculo-cutaneous
External saphenous
Anterior tibial

PLATE V.

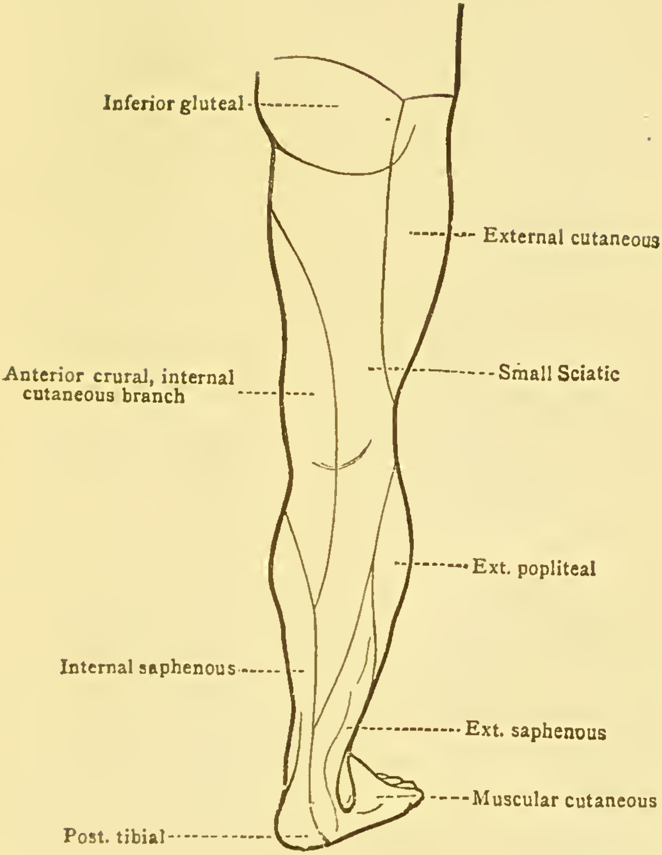

Inferior gluteal
External cutaneous
Anterior crural, internal cutaneous branch
Small Sciatic
Ext. popliteal
Internal saphenous
Ext. saphenous
Muscular cutaneous
Post. tibial

PLATE VI.

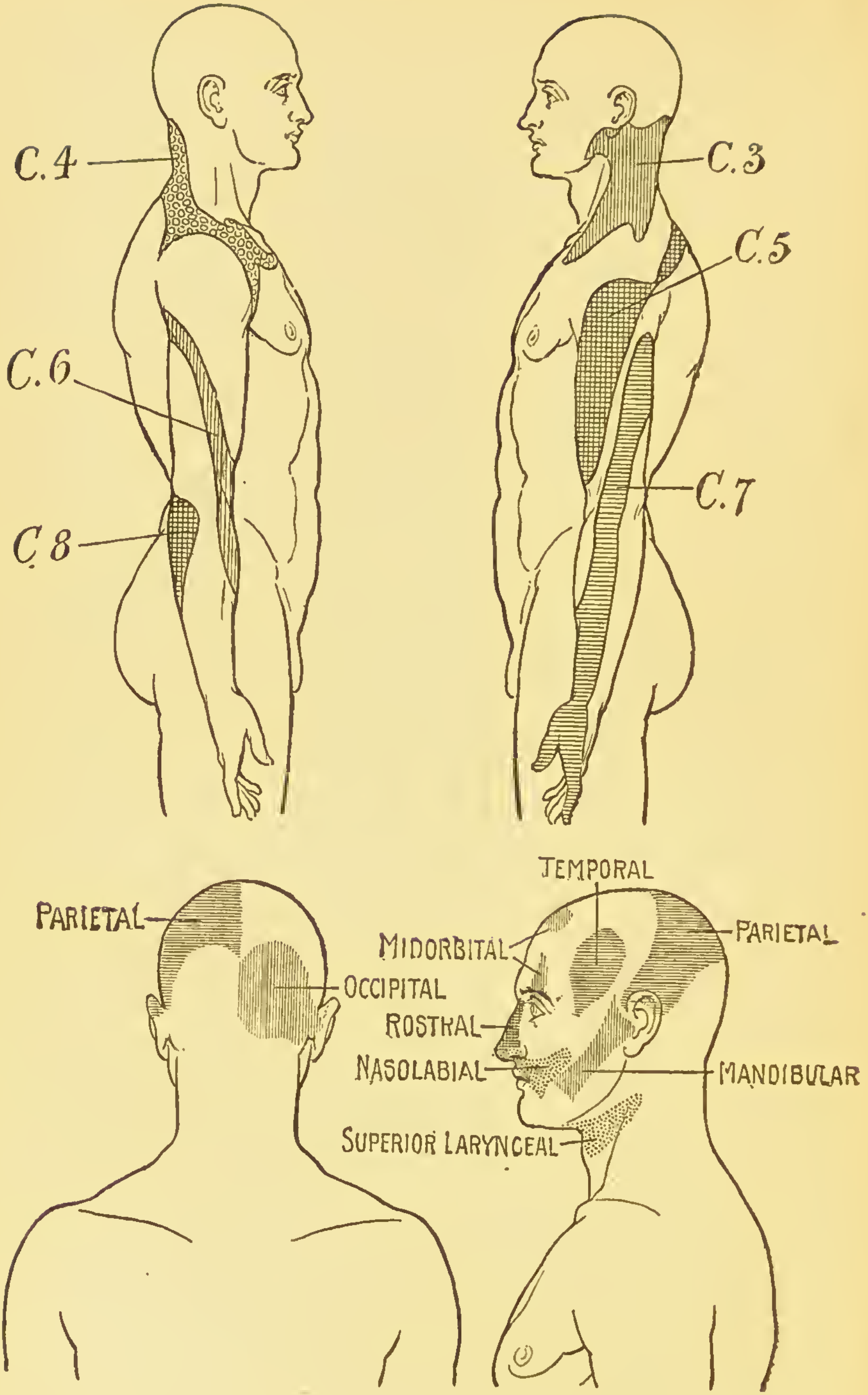
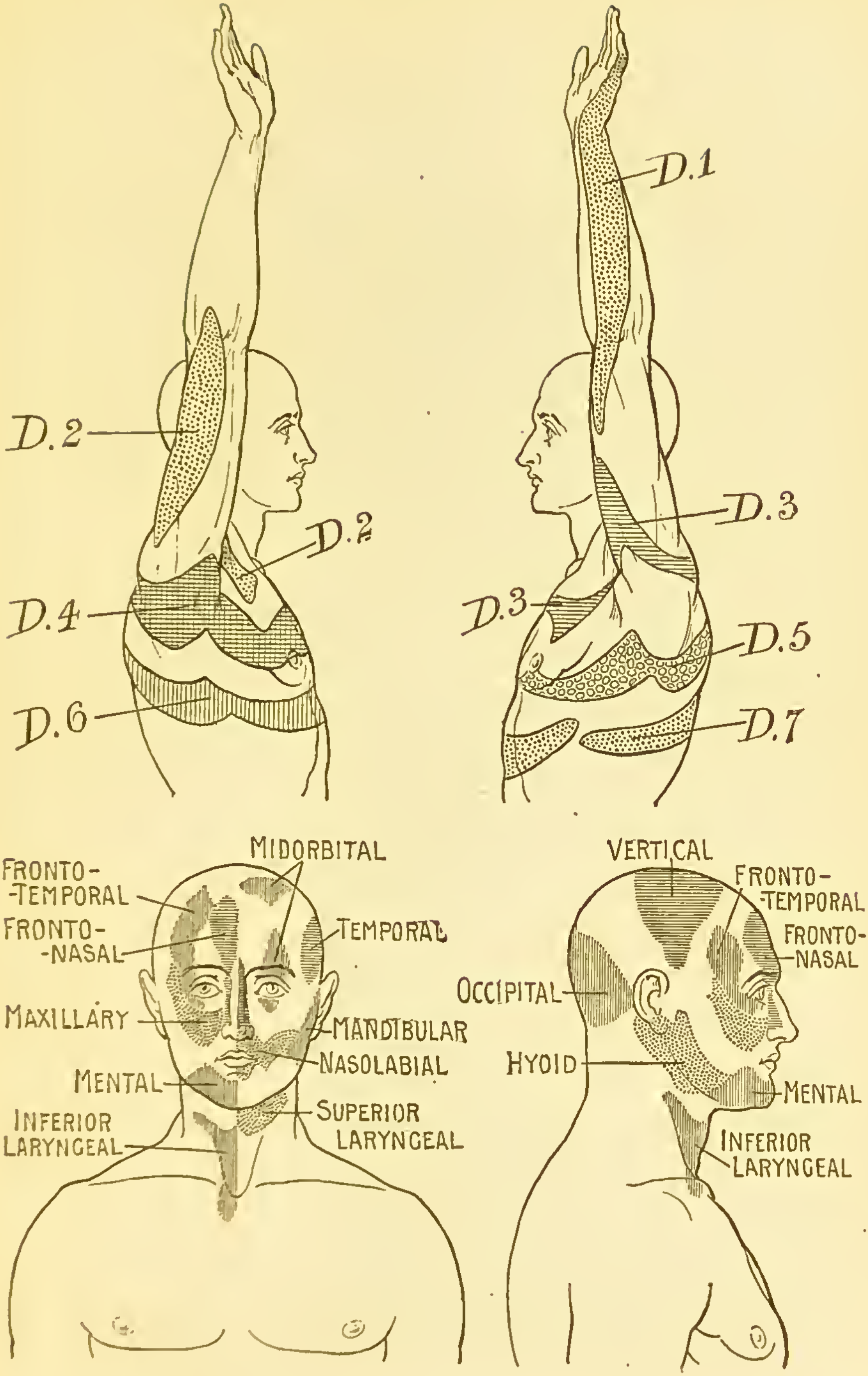
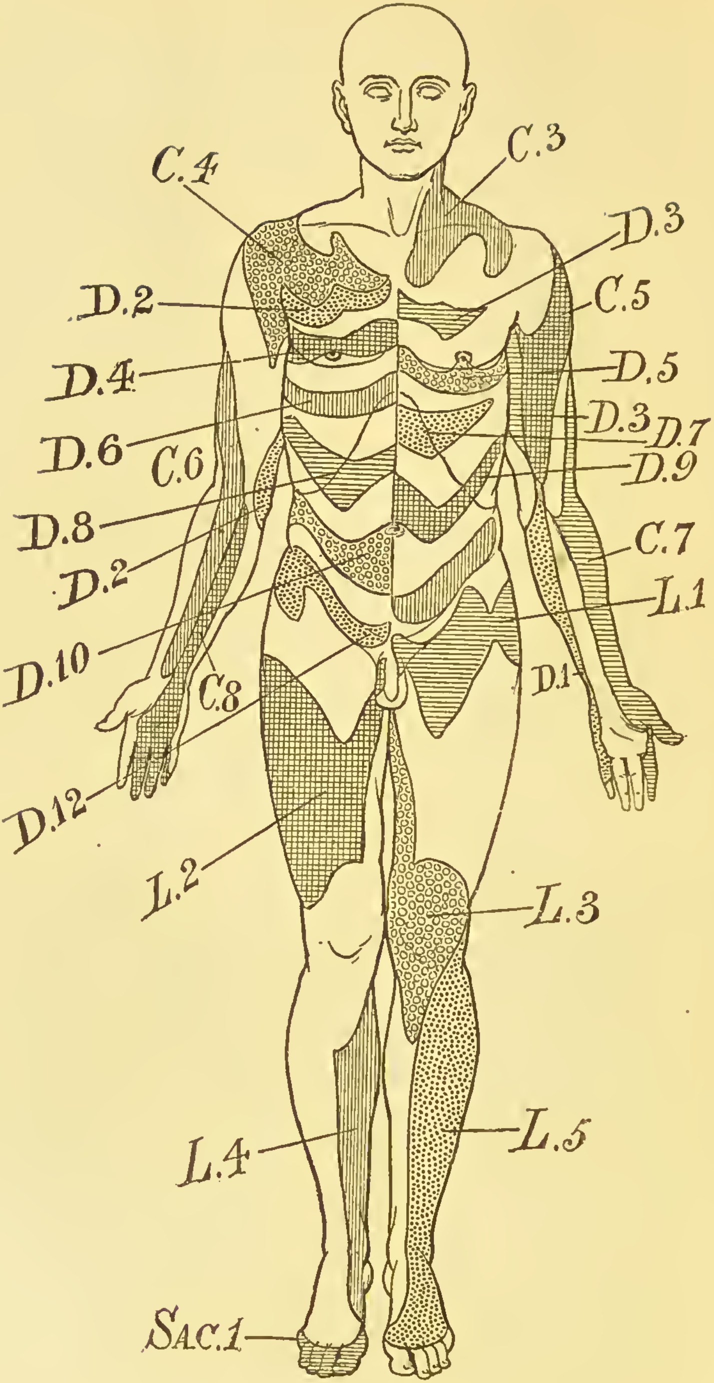
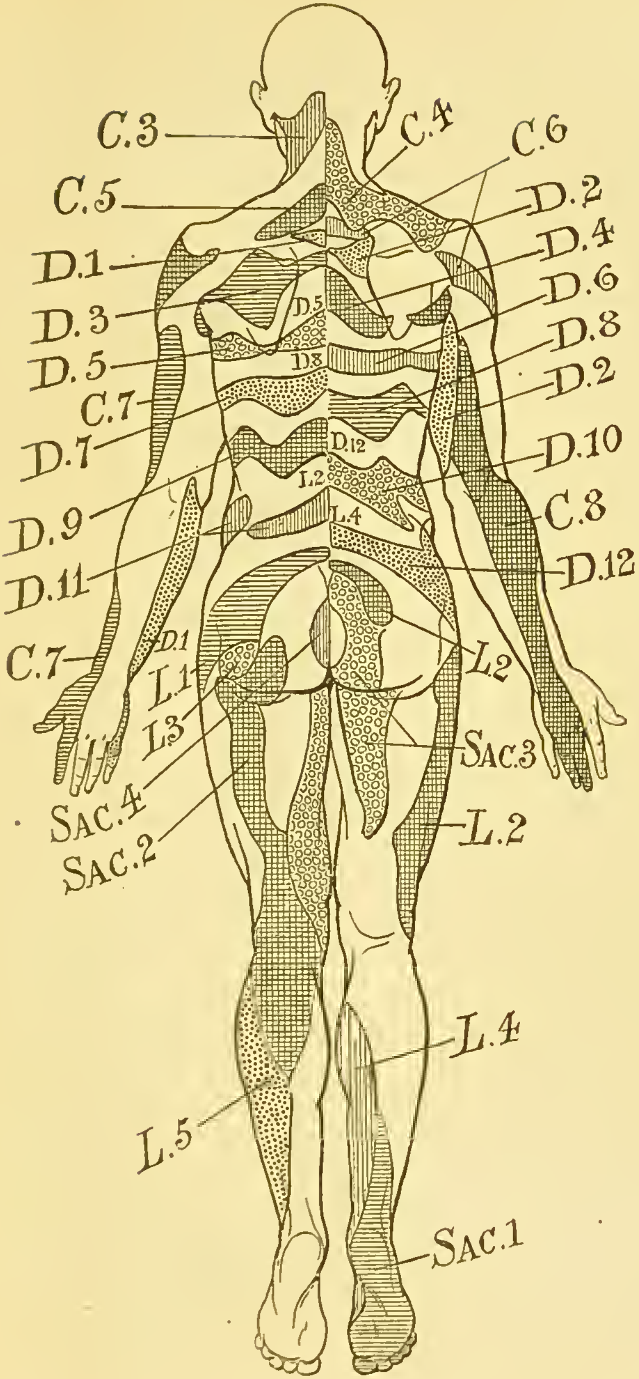

C.4
C.3
C.5
C.6
C.7
C.8
TEMPORAL
PARIETAL
MIDORBITAL
PARIETAL
OCCIPITAL
ROSTRAL
NASOLABIAL
MANDIBULAR
SUPERIOR LARYNGEAL

PLATE VII.


D.2
D.2
D.4
D.6
D.1
D.3
D.3
D.5
D.7
MIDORBITAL
FRONTO-
-TEMPORAL
FRONTO-
-NASAL
TEMPORAL
MAXILLARY
MANDIBULAR
NASOLABIAL
MENTAL
INFERIOR
LARYNGEAL
SUPERIOR
LARYNGEAL
VERTICAL
FRONTO-
-TEMPORAL
FRONTO-
-NASAL
OCCIPITAL
HYOID
MENTAL
INFERIOR
LARYNGEAL


C.4
C.3
D.3
D.2
C.5
D.4
D.5
D.6
D.3
D.7
C.6
D.9
D.8
C.7
D.2
L.1
D.10
C.8
D.1
D.12
L.2
L.3
L.4
L.5
SAC.1


C.3
C.4
C.6
C.5
D.2
D.4
D.1
D.6
D.3
D.5
D.5
D.8
D.8
D.5
C.7
D.2
D.7
D.12
D.10
L.2
D.9
L.4
C.8
D.11
D.12
D.1
C.7
L.1
L.2
L3
SAC.3
SAC.4
L.2
SAC.2
L.4
L.5
SAC.1

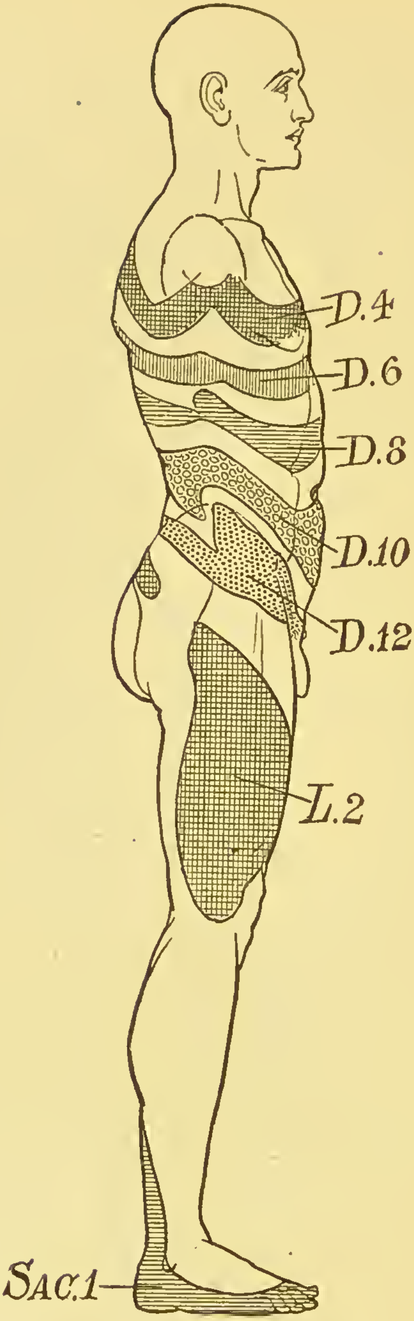

D.4
D.6
D.8
D.10
D.12
L.2
SAC.1

PLATE XI.

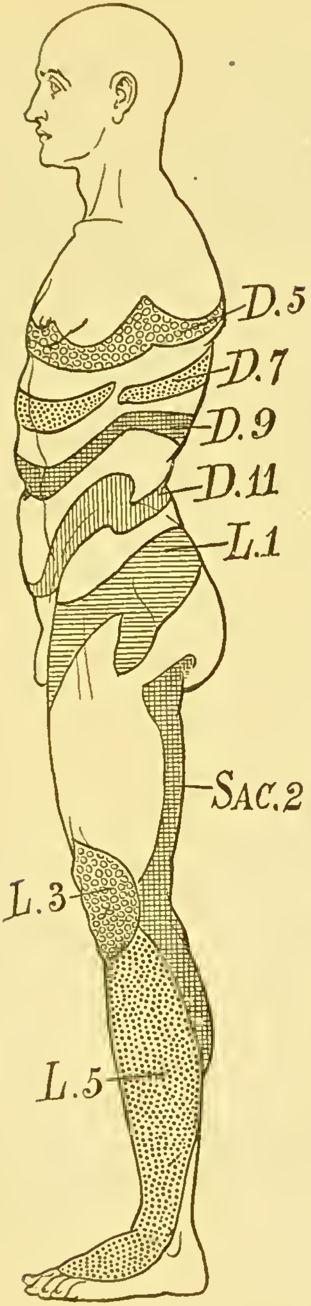
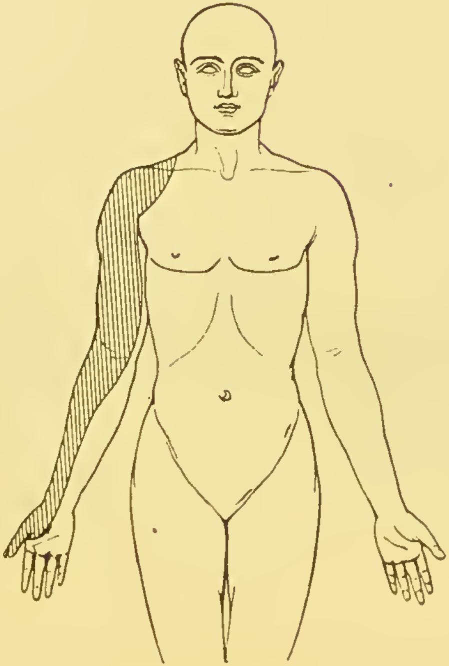


PLATE XII.



FIG, 1.

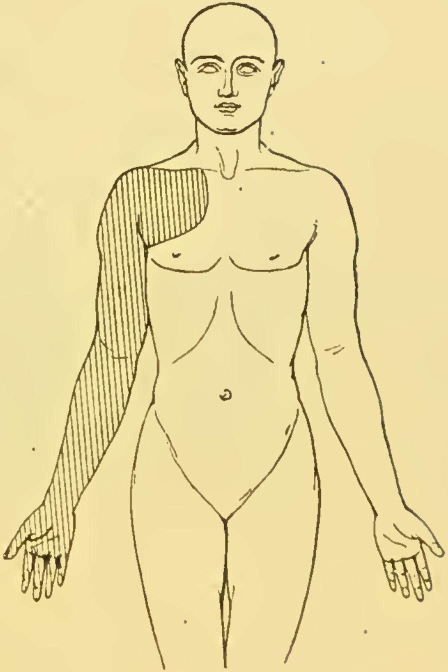


FIG. 2.

PLATE XIII.

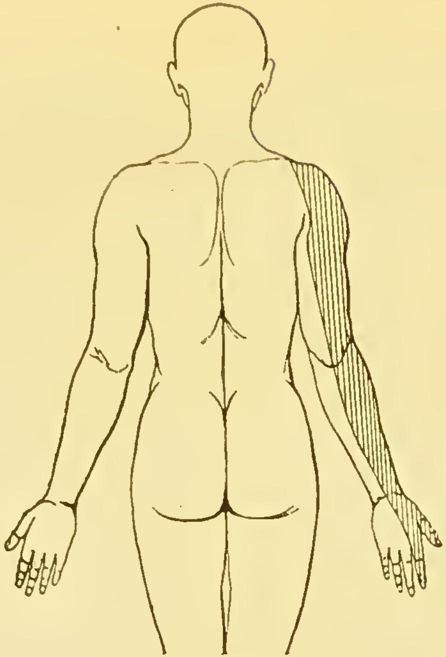


FIG. 3.

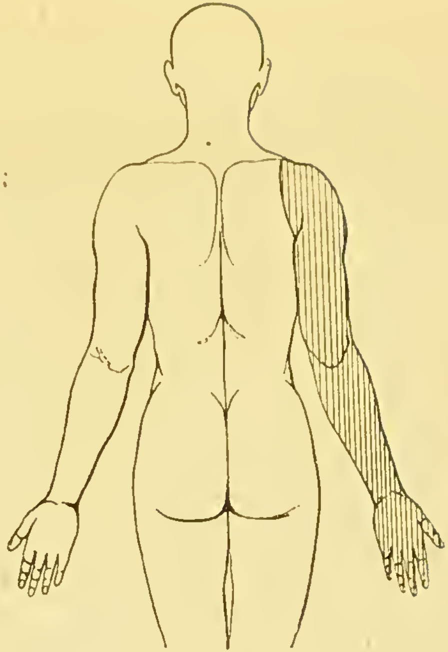


FIG. 4.

Diagrams of loss of sensation from lesions of the cervical roots and of the "so-called" motor cortex.
Figs. 1 and 3 are from a lesion of probably the fifth, sixth, and seventh cervical roots and part of fourth.
Figs. 2 and 4 are from a case where the whole of the "upper limb area" of the motor cortex was removed by Mr. Horsley.

Relations between the superficial origins of the spinal nerves from the spinal cord and the spinous processes of the vertebræ (after Reid).

Superficial Origins of Spinal Nerve Roots.	Apices of Spinous Processes.
Cervical 1	
2	1 Cervical
3	
	2
4	
	3
5	
	4
6	5
7	
	6
8	
Dorsal 1	7
2	
	1 Dorsal
3	
4	2
5	3
6	4
7	
	5
8	
9	6
10	7
11	8
12	
	9
Lumbar 1	10
2	
3	11
4	
5	
Sacral 1	
	12
2	
3	
4	
5	
C 1	
	1 Lumbar

TABLE

Giving the groups of muscles supplied by the spinal cord, and the relation of the reflex actions to it, at the levels corresponding to the roots of the different spinal nerves.

	Spinal Cord at Level of	Muscles.			Reflex.	
Small rotators of head	C. 1	Small flexors of head				
Complexus, Splenius	2	Depressors of hyoid bone	Sterno-mastoid			
	3	Levator anguli scapulæ				
		Scaleni				
		Trapezius				
	4	Diaphragm (? 3rd and 5th C.)				
	5	Levator anguli scapulæ, Scaleni		5		
		Supra-spinatus, Rhomboids, Subclavius				
		Infra-spinatus				
		Teres minor				
		Biceps and Brachialis anticus				
		Deltoid				
		Supinator longus			Radius tap	
		Supinator brevis (?)				
		Pectoralis major (clavicular part)				
Erector spinæ from the whole length of spinal cord		Serratus magnus				
	6	Subscapularis (? 5th C.)		6	Scapular	
		Pronators				
		Teres major				
		Latissimus dorsi (? 7th C.)				
		Pectoralis major (sternal part) (? 7th C.)				
		Triceps (? 7th C.)			Triceps tap	
		Serratus magnus (? 7th C.)				
	7	Extensors of the wrist and digits		7		
	8	Flexors of the wrist and digits		8		
	D. 1	Interossei and small muscles of hand		1		
	2	Dilator fibres of pupil	Intercostales	2	Pupil dilatation to shade	
	3			3		
	4			4	Epigastric	
	5	Rectus abdominis		5		
	6			6		
	7			7		
	8			8	Abdominal	
	9	Obliquus externus		9		
	10	„ internus		10		
	11	Transversalis		11		
	12			12		
	L. 1	Quadratus lumborum		1	Cremasteric	Vesical
	2	Cremaster		2		
	3	Sartorius		3	Knee jerk	
		Adductors of hip				
		Ilio-psoas				
	4	Extensor quadriceps cruris		4		
		Abductors of hip				
	5	Flexors of knee		5	Ankle clonus	Vesical
	S. 1	Calf muscles		1	Plantar	
	2	Glutei		2	Gluteal	Anal (? 4th S.)
		Peronei				
		Anterior tibial muscles				
		Intrinsic muscles of foot				
	3	Perinæal muscles		3		
	4	Perinæal muscles and rectum (?)		4		

In the above table the list of the muscles for the limbs is that given by Thorburn (*Surgery of the Spinal Cord*, 1889); and though it differs from the lists for the brachial and lumbar plexuses made on anatomical grounds, such as Herringham's, it is most in accordance with the clinical cases. As to whether the group of muscles supplied by a root is the same as the group supplied by the spinal cord at the level of that root cannot be answered definitely. The two are often assumed to be identical; but I think that this is probably not the case. The figures in brackets mean that the muscle is sometimes supplied by other roots—*e.g.* the diaphragm is sometimes supplied by the 3rd or 5th cervical. The vesical and anal reflexes and the knee jerk are transferred from Sherrington's work on the monkey.

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